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THE PHILOSOPHY OF THE OLDER TESTS OF HEARING.*

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In order to justify its existence every procedure in scientific or clinical work should, so far as possible, have a definite purpose and show certain results; in other words, it should have a raison d'etre. It seems to me that this idea applies likewise to the functional examination of hearing, and I will endeavor to sketch the basic principles underlying the theory of the older tests, point out their practical application, and their bearing upon the interpretation of the clinical findings. The subject of audiometric measurements, as well as one of the modifications of the Rinné test will be discussed by Dr. E. P. Fowler, so we will merely mention them briefly later on.

The purpose of functional testing of hearing is twofold:

1. To determine the presence of impaired hearing, if any, and the degree thereof.

2. To decide upon the location of the hearing defect.

Various methods, including the use of the voice, acoumeter, watch, Koenig rods, tuning forks, resonators, whistles, monochord and audiometers are employed in the examination of the hearing function.

Observation of the patient is an important factor and much may be learned from it regarding the state of the individual's hearing even before the tests are begun. For instance, the loudness of the voice may be utilized in differentiating between the impairment of the

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conduction and perception apparatus. When there is a decided middle ear deafness, for instance an otosclerosis or a chronic tubal catarrh, the patient has a marked autophonia, his own voice appears very loud to him, and believing that it likewise sounds very loud to others, he tends to speak very low. On the other hand, when there is a pronounced change in the perception apparatus the bone conduction is very materially diminished, his own voice sounds very feeble to him, and in order to make others understand he is likely to talk very loud. When there is a marked change in the hearing there is often noted a peculiar attitude of the head, the patient inclining his better ear toward the speaker, and often using the time-honored method of placing the hand behind and in contact with the auricle. Dr. Paul E. Sabine, of Riverbank Laboratories, concludes from his experiments that the hand really serves as well as a collector of sound and as a resonator as do most mechanical appliances, except the very large ear trumpets or horns. One often notices that those who have impaired hearing, watch very closely the lips of the speaker, thus consciously or unconsciously doing a great deal of lip-reading. The patient's occupation may have an important bearing on the lesion which is present. For instance: nerve degeneration is frequently seen in boilermakers or others who work in the presence of much noise.

Examination of the ear itself is very important. A good otoscopic examination includes a careful inspection of the auricle and external auditory canal for cerumen, inflammatory conditions, tumors, malformations, etc. Then note the appearance of the drum membrane, its color, luster, mobility; see if pathological changes, such as hyperemia, retraction, thickening, scars or perforations are present. If a large perforation exists observe the condition of the tympanic mucosa. The nose, nasopharynx and pharynx should be examined for the presence of any abnormalities which may have influence on the ear, especially by way of the Eustachian tube. We believe too much emphasis should, however, not be placed upon the presence of septal deviations and others forms of nasal obstructions as the cause of ear involvement; nor should these alone serve as indications for intranasal surgery unless the obstruction to breathing, or a decided interference with drainage of the nasal accessory sinuses call for relief *per se*. If it is shown that the inner ear is involved, operation on the nose will surely not aid hearing. Only where there is direct interference with aeration of the Eustachian tube by very large tonsils, conditions in the nasopharynx, such as tumors, large adenoids or hypertrophied posterior ends of inferior turbinates, etc., do I believe we may expect amelioration of the middle ear process by operative work in the nose, pharynx or nasopharynx.

N. B.—The personal equation of examiner and patient, *i.e.*, whether he is a willing one, and tries to hear too much, or a malingerer who pretends to hear very little, has an important bearing on the results of the functional examination.

FUNCTIONAL TESTING.

The Politzer Acoumeter: This little applance is still used by many, the patient being at one end of the room with one ear occluded by the finger, and the eyes averted or closed. The examiner, while clicking the acoumeter, approaches the patient until the distance of hearing in the examined ear is noted. The fallacies of this test are, first, that the acoumeter is rather high pitched, and with it only one pitch level can be used. The patient, for instance, who has a conduction apparatus impairment will hear the high pitched acoumeter even though he has defective hearing for the lower tones. Secondly, it produces a noise and not a pure tone.

The ordinary pocket watch is used by some, the manner of testing being similar to that with the acoumeter in approaching the ear, etc., but the fallacies are that here also many noises are produced instead of a pure tone, and, secondly, that since no two watches are exactly alike in their pitches or intensity, no definite criterion for comparison with other men's tests may be had.

Tone is sound due to a number of equal regular vibrations; a noise is sound due to irregular vibrations, irregular both in length and in time.

Tones are best obtained by tuning forks, since they give a purer tone and have a decreasing strength of sound after being struck so one can judge the hearing acuity. *2.* Noises are used in testing as produced by the watch and Politzer acoumeter. *3.* Mixed tones and noises are produced by the voice.

The voice in the form of unaccentuated whisper or unaccentuated conversation is one of the most frequently used tests. The voice cannot be standardized as we would like to have it, nevertheless from a practical viewpoint it is perhaps the most important test, since the criterion of proper hearing is, after all the ability to understand the speech of our fellowmen. In order to get as nearly a uniform result as posible, the examiner should drill himself into the habit of using the voice with approximately the same intensity and pitch each time, and this is best accomplished by employing the residual air. Use high pitched and low pitched words or numbers, combining the two as, for instance, 27, which includes both a high and a low pitched sound, or 48, which has low pitched ones, or 76, which has two high pitched sounds. Note the distance at which the low, the high, or both are heard. In the small rooms in which most otologists in the

larger cities must carry out their examinations it is better to test with the whispered voice; if that is not heard one can use the conversation tone.

Fallacies of Testing with the Voice: It is very important that the individual be tested with his eyes closed or averted from the speaker, that the opposite ear be well occluded with a moistened finger, cotton plug or noise apparatus and that no part of the body come in contact with the wall or other fixed structures in the room excepting the floor, otherwise some of the sounds may be carried via the bones of the arm and the body to the ear so that hearing will not be entirely by air conduction. We should remember that with the voice tests some of the sound waves strike the skull and are transmitted to the ear by bone conduction, though the vast majority of sound reaches the inner ear by way of air conduction through the external auditory canal.

It is very important when using the voice, the watch, the acoumeter, or other methods whereby one determines the hearing distance, to approach the patient from afar to the point at which he first begins to hear; and not to produce the sound close to the ear and then move away from him, because it is a psychological fact that as we recede from the person he will, from memory, often say that he still hears the sound when he no longer does.

It is very difficult to completely occlude the opposite ear, but every effort should be made so that the patient hears the sound only in the ear that is being tested. In order to overcome this difficulty a 2 or 3-meter speaking tube may be used, the tip of one end being inserted into the patient's ear, the examiner whispering through the other funnel-shaped end. The sound is then carried only through the lumen of the tube to the examined ear, and if repeated one is certain the hearing has been by way of the ear that is being tested.

After testing with the voice, watch or acoumeter, the ears may be inflated with the Politzer bag, or preferably by way of the Eustachian catheter, and the degree of hearing again determined. The presence or absence of an improvement in hearing following careful inflation is an important index in the determination of the prognosis. If a decided improvement is observed it usually means that further treatment will benefit the patient. If, on the other hand, no improvement whatever is noted (especially after inflation has been repeated two or three times on different days) experience leads one to feel in most cases that local treatment will be of very little avail.

The Use of Tuning Forks: These have proven of immense value in functional testing, so it will perhaps not be amiss to mention a few of the characteristic properties of forks. The prongs move in trans-

verse vibrations of wide amplitude but slight intensity, while the stem moves in longitudinal vibrations of small amplitude but high intensity for the same length of time. Forks when sounding have a certain definite decrement of vibration so that the intensity rapidly diminishes after the first few seconds, then more slowly in a long curve of geometric progression. This decrement is different in various forks of the same pitch, but is practically the same for any one fork unless there is a great variation in the intensity with which the fork is struck or excited. Every fork has a certain fundamental tone and then a considerable number of overtones or harmonics. In order to remove the overtones and bring out only the fundamental tones, some forks are provided with clamps, weights or heavy rubber bands on the prongs. Their disadvantage lies, however, in the fact that they diminish the time for which the fork will vibrate. Edelmann found, however, that it was possible to eliminate overtones (and especially the first one, which is usually the most pronounced), by so forging the fork that the first overtone and the so-called "plate tone" have the same pitch. When a forking is lying on wooden ledges and is struck at certain points the so-called "plate tone" is produced, as contrasted with the sound emitted when the fork is excited in the ordinary manner. If my any chance the "plate tone" and the fundamental prong tone happen to be of the same pitch, the fork will be entirely silent.

We believe that some of the requisites which good forks should have are:

1. They should be of one piece of metal.

2. The handle should be long enough so it can be easily, yet loosely, held without damping or stopping the vibrations.

3. The fork should sound for a long time.

4. There should be weights for the lower pitched forks in order to remove the overtones.

5. Forks should not be nickeled, for when the nickeling peels adventitious sounds are produced by the loose pieces.

6. Rustless metal if feasible should be used to prevent changes in pitch by oxidation.

Forks as usually employed range in pitch from C^2 (16 d. v.) to c^6 (8192 d. v.). A 12-double vibration fork is made, but it is very large and difficult to manipulate. Forks higher than c^6 have also been constructed, but not for general otologic practice.

Methods of Exciting Forks: The general rule is that the lower the pitch of the fork the softer should be the material used for striking or exciting it. For the very lowest ones the side of the hand (hyponethenar eminence) may be used; for those of medium

pitch, a rubber-covered pleximeter, and for the highest forks a metal hammer. In order to obtain a uniform excitation we have followed a definite routine; we start some of the lower forks, like the unweighted a (108 d. v.), by allowing them to fall from a perpendicular position to a horizontal one, striking the knee. The medium pitched forks, like the small a-1 (435 d. v.), we usually hold at right angles to the body with the broad surface of one of the prongs uppermost, allowing a small pleximeter (such as is used by neurologists for eliciting reflexes) to fall of its own weight from a perpendicular to a horizontal position, striking the prongs. For the highest pitched fork three great degrees of excitation may be employed; first, lightly rubbing the prongs with the fingers; second, if the sound is not heard striking one of the prongs with the finger nail; and third, if still not heard, striking it with a metal hammer.

It is probably best to strike the forks at the "percussion" point at about the junction of the distal and middle thirds of the prong, since this gives the greatest intensity with the least overtones.

In order to determine which part of the tuning fork prong gives forth the greatest intensity of sound and thus have a basis for deciding which surface or portion should be presented to the ear in routine testing, I studied with the Webster Phonometer the sound emitted by the different parts of the tuning fork prongs. The findings were embodied in a paper read before the American Otological Society in 1924. Tests were made with electrically-driven fork, with fork actuated by means of a pendulum weight, with forks held before Schaefer resonators, and the results carefully noted by means of the very delicate and accurate Webster Phonometer, which measures intensity of sound. Furthermore, the degrees of intensity were compared by presenting the prongs to the ears of a number of individuals in a rather long series of experiments. In this manner we found that the broad or outer surface of one of the prongs showed uniformly the greater intensity of sound. This could *a priori* be expected from the fact that it is from this surface that the sound waves are emitted, this being the only surface that really exerts pressure against the air. The narrow surfaces of the prongs usually give an intensity next to that of the broad surface and usually greater than the ends of the prongs. The angle between the two surfaces of the prongs showed the least intensity, due to the fact that at this point there is interference of sound waves. From a practical standpoint I believe it is best to hold the broad or outer surface of one of the prongs of the fork parallel to and as close as possible to the ear without touching either the auricle or vibrissae in order to get the greatest intensity of sound. However, if the same method is used in

every case it will probably not make much difference whether the narrow surface or the ends of the prongs are held at uniform distance from the ear, but the edges of the prongs should not be presented. I have always felt that in clinical practice a uniform method should be employed in order that the results obtained from examination may be comparable one with the other. From a scientific standpoint we should seek to develop the highest efficiency of any apparatus, and so present it to the ear so that the maximum sound is appreciated. For that reason we believe that the broad or outer surface of the prong should be presented to the ear.

Testing Range of Hearing—Lower and Upper Tone Limits: Begin with the lowest fork at hand and have the patient close his eyes so that he will not be influenced by the sight of the fork or its proximity to the ear, and then have him state when he hears a certain sound. Do not ask him, "Do you hear this," for the tendency is to say yes; but have him tell when he first appreciates the tone. If he does not hear the lowest forks proceed up the scale until you find the point at which he begins to hear. After that continue to present the forks in ascending tones or octaves until the highest points are reached, closing the opposite ear.

Two important precautions to be observed are: that the patient must be instructed to distinguish between actually hearing the very low tones, and merely feeling the impact of the sound waves against the auricle. In the second place, when using the higher pitched tones it is necessary that the opposite ear be well occluded so that the sound waves are not carried over and appreciated by the ear which is not being examined at the time. It is practically impossible to entirely exclude the very highest tones from the opposite ear, especially when the fork is struck with a metal hammer.

The upper tone limit of hearing is further tested by means of whistles, such as the Galton, the modified Edelmann-Galton, or the more recent Schaefer-Galton whistles. These whistles are supposed to have an upper limit of about 20,000 or 22,000 double vibrations. It is important to remember that with whistles one can test sound only by way of air conduction.

The difficulties or fallacies connected with testing by means of the whistle are several. In the first place, it is hard for an individual to distinguish between the blowing sound and the actual high pitched whistle of the highest tones. In the second place, there is difficulty in obtaining uniform pressure of air unless certain special devices are used. Lastly, in the handling of the whistles unless great care is exercised rusting or other impairment of the fine edge of the whistle occurs and causes a decided change in the pitch of the tone. We

believe that the Schaefer-Galton modification possesses certain advantages over the other types in that: *1.* The aperture is fixed at the constant point and thus needs no adjusting. *2.* That it requires only one hand to manipulate; and, *3.* it is considerably cheaper than the Edelmann-Galton whistle.

One of the most reliable instruments for the testing of the high tones is the monochord, and the Schaefer-Struycken model is probably the best one for this purpose. The monochord is much more dependable than the whistle and less easily disarranged. By means of the monochord the high tones can be tested both by air and by bone conduction, and it is a fact that with this instrument the very highest tones are better appreciated by bone conduction than by air. The lower tones on the monochord are evoked by transverse vibrations when bowing the wire with a violin bow. The higher tones are longitudinal and are produced by rubbing the wire with a pad of cotton, etc., moistened with turpentine or alcohol. When bone conduction is being tested that end of the monochord which has an ivory knob is held against the mastoid process or some other part of the skull.

The usual opinion is that high tones are better heard by air conduction than by bone. With the mononchord. however, the very highest tones are better appreciated by bone as previously stated. Kalaehne says that with the monochord the high tones are better heard by bone because the amplitude employed is the same for air and bone conduction. With tuning forks this is not possible, because the stem vibrations practically always have an amplitude of only about 1/100 of that of the prong. He further believes that with the lowering of the upper tone limit the bone conduction is relatively more diminished than is the air conduction.

For testing the complete range of tones from the lowest to the highest, Koenig rods are employed by many as adjuncts to tuning forks, monochords, etc.

Bone and Air Conduction: Sound is conducted to the ears by both bone and air conduction. Transmission by air is through the external auditory meatus to the tympanic membrane and then by way of the ossicular chain to the internal ear, or by way of the nose, pharynx and Eustachian tube, to the middle ear and then to the internal ear. Bone conduction also has two pathways, that is to say, the craniolabyrinthine, through the cranial bones directly to the petrous portion of the temporal bone and inner ear; or the craniotympanic through the cranial bones to the middle ear and then to the inner ear. According to Bezold the difference in mechanism of the air and bone conduction lies in the fact that by way of air sound waves impinge upon the flat surface of the drum membrane, and by

way of bone they strike the edge thereof. Usually air conduction is very much longer than bone conduction. Bezold states that the function of the conduction apparatus consists in transforming the longitudinal air waves (including those which directly strike the skull) into transverse vibrations of this mechanism (as a whole together with the column of labyrinthine fluid). The ossicular chain is most essential in transmitting the lower tones of the musical scale (tones produced by vibrations of large amplitude but little force), which the ossicular levers convert into vibrations of smaller amplitude but of greater intensity. The higher tones, however, are produced by vibrations of smaller amplitude, whose relatively greater intensity is capable of producing a wave in the labyrinth fluid without increase in force by means of the leverage system of the ossicles.

The Weber Test: This test is used for determining the presence of lateralization of sound. As a rule, one of the heavier low pitched forks is used for this purpose. The A (108 d. v.) fork is the one we are in the habit of employing. After striking it the fork is placed on the median line of the vertex, or as some do, at the root of the nose, on the teeth or the chin. When applied to one of the latter positions the tone is usually heard louder because the mouth and nasopharynx act as resonators. The patient is then asked whether he hears the sound of the fork louder in the head itself or in one of the ears.

Interpretation of This Test: Normally with both ears in the same condition the sound is usually appreciated "in the head," as the patient expresses it. When there is a conduction apparatus impairment the sound is usually heard in the worse ear, but if an inner ear or nerve involvement is present the sound is usually heard in the better ear. There are, however, many exceptions to these rules. In a series of tests made by myself many years ago in which the A (108 d. v.) fork, the a1 (435 d. v.) and the weighted c^1 (154 d. v.) forks were employed, it was found that in 16 to 17 per cent of the cases the Weber was lateralized to the side contrary to the one expected from the nature of the lesion. In most cases (85 to 89 per cent, depending upon the fork used) the Weber was the same with the forks on either the vertex or the forehead. In only 10 to 15 per cent did the position make a difference in the lateralization.

The degree of lateralization may be noted by shifting the fork away from the median line of the head. For instance, if the sound is heard in the left ear with the fork held in the median line, shift it somewhat toward the right side of the head and see how far from the median line the fork can be moved without influencing the lateralization to the left ear. If heard in the left ear with the fork far

over on the right side there is very marked conduction impairment in the left ear, or a very decided nerve change in the right ear. In this connection we must remember that if a tuning fork is placed laterally to the median line of the head diagonal resonance or crossed perception of sound is obtained. With the fork in the median line the head is divided into two equal portions, both of which apparently resonate equally well, but when the fork is shifted to one side of the median line, the skull is divided into two unequal resonating masses, the smaller of which is the one on which the stem of the fork is resting, and the larger is the contralateral side. For instance, if the fork is placed external to the median line on the right side with normal individuals the sound is heard louder on the left side than on the right, and vice versa.

The Weber test is often of considerable aid in differentiation between external otitis and an otitis media, especially in young children. Where there is considerable difficulty in otoscopic examination lateralization of the Weber is of considerable aid in differential diagnosis. We usually have lateralization with the otitis media, but not as a rule with otitis externa.

A most important phenomenon is the change of the lateralization from one side of the head to the other. If, for instance, during the course of a left acute otitis media (which is a conduction apparatus impairment) with the Weber heard in that ear there is suddenly a change in the lateralization to the opposite or right ear, it usually means that there has been an extension of the process with involvement of the inner ear (labyrinthitis in the left ear), and then the Weber is heard in the opposite (right ear), or better ear. The change in the lateralization is thus very significant of an extension of the pathological process from the middle ear.

Schwabach Test: This test is employed for determining the duration of the bone conduction of the individual as compared with the normal standard. While it is often believed that this test is always done with the fork on the midline of the vertex as in the Weber test, many otologists place the fork either on the vertex or the mastoid process, or both. In this country Randall and others employ the Gardner-Brown modification with the fork placed at the root of the nose and the head bent backward. Various men employ forks for this test ranging all the way from C (64 d. v.) to c2 (512 d. v.).

We believe that a rather low pitched heavy fork, Bezold a unweighted (108 d. v.) is well suited for this test. After the proper excitation of the fork it is placed in the median line of the vertex. Care should be taken that the hairs are separated so that the fork rests directly upon the scalp. Secondly, the patient should be cau-

tioned against confusing the feeling of the vibration of the fork with the hearing of the actual tone thereof. Third, the fork should be allowed to rest, if possible, of its own weight, since the pressure with which it is applied changes the amount of the bone conduction. Fourth, the stem of the fork should not be held too tight as the vibration is thereby damped or stopped. When the patient states that he no longer hears a sound, remove the fork for a second because of the so-called "fatigue" symptom, then replace it upon the head and note whether it is again heard. When no longer perceived the fork is set upon the head of the examiner, or some other observer whose bone conduction is known to be normal. In order to make this test more objective, nad not dependent upon some other individual for comparison, we have been of the habit of finding out what is the average duration of hearing for the particular fork employed in a great number of normal individuals. Having determined this figure we simply compare it with the length of time that the fork is heard by the patient, and thus see at once whether the bone conduction is lengthened, shortened or normal.

The duration of bone conduction even in normal persons varies considerably, depending upon the age (at or after middle age it is usually considerably diminished), although it may differ in persons of the same age. The thickness of the cranial bones, the size of the air spaces, such as the mastoid cells, as well as certain anomalies of the skull, such as traumatisms, adhesions, etc., between the dura and the bone, etc., influence the Schwabach by giving a greatly shortened bone conduction in spite of the normal hearing by air conduction. The amount of the hair, the tension of the skin, the contact with the auricle, and the pressure with which the fork is applied, all these may cause variations in the bone conduction. It is, however, necessary to remember that a slight increase or a slight diminution in the duration of bone conduction does not have any definite significance. Politzer states that from a prognostic point of view cases with lengthened bone conduction are usually more favorable in their course as the result of the treatment than where it is shortened. But we must not forget that a typical otosclerosis which usually shows prolonged bone conduction has a very poor prognosis. On general principles we may say, bearing in mind the various factors which have been previously mentioned, that lengthened bone conduction usually means impairment of the conduction apparatus, and a diminished bone conduction usually spells involvement of the inner ear or auditory nerve.

Oscar Beck, of Vienna, called attention to the diminution in bone conduction at times noted in cases of lues with good hearing and no aural symptoms. He says that this lowered bone conduction may be

found in 80 per cent of all syphilitic subjects, and that it appears mostly in the second, and only rarely in the first stages. We have found this strikingly illustrated in a number of cases of rather early lues where the hearing apparently was still very good but bone conduction markedly diminished and the Wassermann found strongly positive.

From a careful study some years ago of the Schwabach test in 100 cases, employing three forks, namely A (108 d. v.), the weighted c^1 (154 d. v.), and the small a1 fork (435 d. v.), I came to these conclusions: *1.* On the vertex the average hearing for the A fork was 48 seconds, for the weighted c^1, 34 seconds, and for the a1, 22 seconds. On the forehead the A fork was heard an average of 47 seconds, the weighted c1 fork, 43 seconds, and the a1 fork, 21 seconds. It is to be note that the weighted c1 fork was heard longer on the forehead than on the vertex, whereas the other forks were heard longer on the vertex despite the presence of thick hair in many of the cases. A grand average of all of the forks showed for the vertex 37 1/10 seconds and for the forehead, 37 3/10 seconds, or practically speaking, no difference at all. The average that the forks were heard on the two mastoids was for the a fork 75 seconds, the weighted c1 fork, 50 seconds, and the a1, 41 seconds, or a grand average for all the forks by way of the mastoids of 60.9 seconds. In this series of cases, the ratio between the hearing by way of the mastoids and that obtained by way of the vertex or the forehead was about three to two.

Rinné Test: This test was first devised by Dr. A. Rinné in 1885 and is employed for comparing air conduction with bone conduction in the same individual. His method was to place the fork on the upper incisors, and when the sound was no longer heard by bone the hearing by air was determined. As it is now performed the test is usually made by setting the vibrating fork upon the mastoid bone in the region of the antrum, and when the sound is no longer heard, holding the prong near the external auditory meatus and observing how much longer the sound is perceived by air. In the normal individual the air conduction is very considerably longer than bone conduction; that is to say, the Rinné is positive. In those cases where bone conduction is longer than air the Rinné is designated as being negative. Since different forks are used by various otologists, one must remember that the lower the pitch of the fork employed, the more likely is the Rinné to be negative, and vice versa. There are at least seven or eight varieties of Rinné reactions.

Bezold considers that air conduction is usually about 30 seconds longer than that by bone in a normal positive Rinné, with the a1

(435 d. v.) fork. When the latter is held only at the meatus without having previously been placed on the mastoid, the duration of air conduction is longer than when the usual Rinné test is done. In fact, it lasts 70 to 80 seconds in many cases. This is due, of course, to the fact that when the stem of the fork is pressed against the bone the vibrations of the instrument are damped and the duration of sounding thereby shortened. When the Rinné test is carried out in the usual manner and the tone of the prongs is no longer heard, if the stem is the inserted into the etxernal auditory canal, the fork is again heard by way of air and usually for a period of about 12 seconds.

Dr. Fowler believes that one obtains a much better idea of the relation of bone to air conduction, in fact derives a real ratio of the one to the other by alternately placing the stem upon the mastoid process and the prongs near the external auditory meatus every few seconds during the test instead of allowing the stem to rest upon the mastoid process until it is no longer heard, and then presenting the prongs to the ear as in the ordinary Rinné test. In this manner there is not so great a damping of the fork as when the stem is held against the bone contrasted with the absence of damping when the prongs are held near the external auditory meatus. (Dr. Fowler will discuss this matter in his paper and I am merely mentioning it here for the sake of completeness.)

As the result of a careful study of the Rinné test in 100 cases I came to the following conclusion:

1. It is probably the most valuable of the tuning fork tests used in the functional examination of hearing. The relation of bone to aid conduction is an important clew to the diagnosis of location of the hearing impairment, at least in many instances.

2. A study of the literature showed that the forks most often used in the Rinné test were the c (128 d. v.), the weighted c^1 (154 d. v.), the unweighted c1 (256 d. v.), and the unweighted a^1 (435 d. v.) fork. In this series of cases the general average in 200 ears with three separate forks, namely, the A (108 d. v.), weighted c^1 (154 d. v.), a1 (435 d. v.) was 44 seconds difference in time between the air and bone conduction. This is longer by far than found by most examiners.

4. While no absolutely definite statement can be made, the unweighted a1 fork (435 d. v.) seems to us the best suited for performing the Rinné test. It has a long period of vibrations, is so constructed that it can be very easily applied to the head or to the external auditory canal, as it has a small rounded knob on the stem, and because it is of such pitch that when testing air conduction the sound is not carried to the opposite ear.

The varieties of the Rinné reaction are:

Two forms of the positive Rinné, namely: *1.* Air conduction greatly exceeding bone conduction giving the normal positive Rinné; *2.* positive Rinné with both air and bone conduction shortened as an inner ear disease.

Three forms of the negative Rinné: *1.* Bone conduction is much increased and air moderately diminished as in the ordinary middle ear disease. *2.* Bone conduction longer than air, but both shorter than normal, so often noted in combined internal and middle ear disease. *3.* Both bone and air conduction greatly diminished. This occurs in far advanced ear disease because air conduction is then lost faster than bone conduction.

Two Forms of So-called "Indifferent Rinné (plus-minus) Rinné:

1. Air and bone conduction are of equal length because air conduction is slightly diminished and the bone conduction is slightly increased. This usually means that a slight middle ear affection is present, and the hearing is quite good.

2. Air and bone conduction equal in duration but both much shorter than normal; an inner disease is usually present and the hearing is very poor.

Infinite Rinné: If not heard at all by air, but heard for a short time by bone we have the "infinitely" negative Rinné. This is very often found in far advanced inner ear disease. In two of our cases we found an infinitely positive Rinné which is very difficult to explain.

The negative "Rinné imaginaire" is sometimes found with one dead labyrinth, and sound really heard in the good ear via bone. Close first one and then the other external meatus to see whether the sound is heard louder by bone.

A good many theoretical objections have been offered to the Rinné test on the grounds that as usually performed, bone conduction is tested by means of the longitudinal vibrations of the stem, while air conduction is determined by means of the transverse vibrations of the prongs. However, since we make the same error in each case, the results may be compared with one another, and that suffices for the practitioner even if it does not for the physicist. In order to overcome these oft-reiterated objections against the use of the fork stem in the testing of bone, and the prongs of the fork in testing air conduction, Dr. John P. Minton and I made some experiments and modified the Rinné test so that the stem of the fork can be used for the determination of both air and bone conduction. After exciting the fork in the usual manner, and placing it upon the mastoid till it is no longer heard by bone, the end of the stem is inserted into a

rubber tubing which has at its other end a hard rubber, olive-shaped, perforated tip which is inserted into the ear. The vibrations of the stem are thus conducted along the lumen of the tube to the ear and heard by air conduction. Physical experiments showed that the sound waves are hardly, if at all transmitted through the wall of the tubing, but are conducted merely along the air column in the lumen. Thus it is very easy to use the stem of the fork for both air and bone conduction, and the various objections, we believe, fall to the ground. The dimensions of the tubing used were: length, 51 c.m., width of lumen, 0.7 c.m., and thickness of walls, 1.5 m.m.

With tuning forks as previously employed one could get a qualitative estimation of the state of hearing, but not a good quantitative one. With the Edelmann-Galton or Schaefer-Galton whistle, and especially with the monochord, the high tones are qualitatively tested. By comparison with a normal standard an objective measure is obtained as mentioned in the discussion of the Schwabach test. However, for the accurate determination of the degree of percentages of loss of hearing, figured in sensation units, more exact methods are necssary, such as properly calibrated audiometers. It seems to me that great credit is due Harvey Fletcher, of the Bell Telephone Laboratories, who has developed the use of the "constant" of damping, or decrement of tuning fork for estimating the loss of hearing in sensation units. This "constant" of damping or decrement is determined by the Bureau of Standards at Washington at a cost of $2.00 per fork. It is thus possible to use forks and obtain a curve the same as with the audiometer. If a good many forks are used in the testing at various pitches this method will consume considerably more time than with the audiometer. On the other hand, if a moderate set of forks is used for this purpose it is much less expensive, more easily manipulated and transported than the audiometer. This is not intended, however, to convey the idea that the good qualities of the audiometer are not appreciated. When using the "constant" the fork is given a standard blow, held near the ear in the usual manner, and when the patient signals that he no longer hears the sound, start the stop-watch. Then place the tuning fork at the examiner's ear and stop the watch when he no longer hears the sound. The reading of the stop-watch represents the difference in the hearing between the patient and the examiner, and this figure multiplied by the "constant" of the fork gives the loss of hearing in sensation units. In order to make this test more objective I believe it is better to find out the average hearing of normal persons by air conduction for the particular fork or forks employed, and then simply noting the time that the patient heard the fork, and multiplying the difference between his

hearing and the normal average. I have used this method and find it very satisfactory.

Gellé Test: This is used for determining the mobility of the foot plate of the stapes. Use a Politzer or other bag connected by way of tubing and an olive-shaped tip with the external auditory meatus. It is essential that the ear piece be so inserted that it is air tight. With the vibrating tuning fork resting on the bulb, vertex, or mastoid, the Politzer bag or bulb is compressed. Normally we have a positive Gellé, in that the hearing of the fork is diminished while the air is compressed in the external auditory meatus. In this manner the foot plate of the stapes is pushed further into the oval window and the hearing is impaired for the time being. When the Gellé is negative, which is the abnormal reaction, the hearing is not diminished by the compression of the air because the foot plate of the stapes is fixed and cannot be moved further towards the labyrinth.

The fallacies of this test are: *1.* that the patients often do not thoroughly understand what is required of them, or *2.* do not pay close attention to the fact that the hearing is somewhat diminished during the time that the bulb is compressed in the normal cases, and *3.* it often happens that the ear piece does not fit tightly into the meatus so that there is an escape of air, and it is then impossible to state whether the foot plate is movable or not. If, however, there is a definite change in the hearing with compression of the air column, it may give valuable information. This is especially true in cases of typical otosclerosis with marked fixation of the stapes foot plate.

Stenger Test for Determining Simulation of Total Unilateral Deafness: It is a well known fact that when the two ears are simultaneously exposed to two sounds of the same pitch and intensity, the one that is produced closer to one ear drowns out, or masks, so to speak, the sound that is entering the other ear. That is to say, if a sound is produced at a certain distance from the right ear the individual cannot know whether there is a similar sound occurring at a greater distance from the left ear (and therefore apparently of less intensity) than in the right ear. It is essential that a pair of forks be employed of exactly the same pitch as it is also best that the patient be blindfolded so that he does not know that two forks are being used at the same time.

We usually employ (following Stenger's suggestion) a pair of Bezold's a1 (435 d. v.) forks. Having struck both forks, one of them is held a certain distance from the left ear, for instance, 10 inches. In the normal individual if a fork of the same pitch is placed less than 10 inches from the opposite right ear, for instance 5 inches,

he will hear only in the ear at which the fork is a shorter distance, namely, the right one, because the sound coming from that short distance drowns out sound entering the other ear at a distance of 10 inches. If, for example, a patient pretends his right ear is deaf, test the left or well ear and find out the distance at which the fork will be heard—let us say 10 inches. In the right ear, which is said to be deaf, the individual will, of course, deny hearing all sounds, no matter how close the fork is approached to that ear. He hears it but denies having any perception of the sound. With one fork kept vibrating, let us say 4 inches from the right ear, and the other fork approaches to within 8 inches of the left ear, the patient will say that he doesn't hear any sound at all, even though he previously admitted hearing the fork with the well ear (that is, the left ear) at a distance of 10 inches. The reason is this: the fork if held in the right ear will drown out the sound of the fork held 8 inches from the left ear. In view of the fact that he claims to be deaf in the right ear, he will say that he hears nothing at all, whereas if he really were deaf in the right ear he would not hear the tuning fork held near that ear but would certainly hear the fork vibrating in the vicinity of the left or well ear. Dr. W. A. Wells, of Washington, recently described a modification of this test which is very simple and ingenious. He uses a piece of rubber tubing of one-quarter-inch caliber, 30 inches in length and a firmness to give a good conduction of sound. In one end of the tube is an ear piece which fits well in the auditory meatus, while into the other end is inserted the stem of a long sounding fork of low pitch (about 120 d. v. per second). After blindfolding, and with the ear piece in the supposedly deaf ear, the vibrating fork is conducted carefully to the meatus of the opposite ear, not too close, but to a point at which it has been previously ascertained that the patient could distinctly hear it; the really deaf person now reports hearing the fork as soon as it comes into the range of hearing of the good ear. The malingerer, unconscious of any hearing in his good ear because of the lateralization of the sound to the alleged deaf ear (the side of the greater intensity), continues to report no hearing and this gives incontestable proof of willful simulation.

There are three other tests that are occasionally used besides those previously mentioned:

1. The Politzer test for determining the patency of the Eustachian tube. With the vibrating fork held before the nose, during the act of swallowing the sound is heard better, provided the Eustachian tube opens properly. The test may be performed with the meati closed so that one hears nothing until there is an act of swallowing. If there is no increase in hearing it shows some interference with the

patency of the Eustachian tube. This test, however, is not of great practical value; Eustachian catheterization gives more definite and dependable information.

2. Bing's test, of which there are two types:

a. In the normal individual, if the etxernal auditory meati are closed with the fingers there is lengthened bone conduction. If there is inner ear disease closing the meati it will also lengthen the bone conduction somewhat. In middle ear disease where the bone conduction is already markedly lengthened it is not increased by thus occluding the meati.

b. Another variety of the Bing test is sometimes called the "entotic" test. A speaking tube is connected with a catheter introduced into the Eustachian tube so that the sound waves enter the tympanum directly. If speech is heard better this way than when the tube is inserted into the external auditory meatus, it means that the interference with sound conduction is not at the stapes, but lies in the tympanic membrane, malleus or incus.

3. Lucae-Dennert Test: This is sometimes used in determining deafness in one ear. It is merely a test of the air conduction; one hears the forks louder with the meatus open than when it is closed. In the test the forks are held near the occluded ear, and after removing the finger one observes whether the hearing is better or not. The theory of the test is, that the worse the hearing the less the difference between hearing with open or with closed meatus; that is, closure of the meatus has less and less influence on the hearing. These three tests are much less important than the ones previously discussed, namely, the Weber, Schwabach, Rinné, Gellé and Stenger. The Weber, Schwabach and Rinné are, I believe, essential in every functional examination, the other five are of service in special instances.

Cranial Resonance: I wish to merely mention the fact that years ago Lucae, Troeltsch, Kessel and many others studied cranial resonance in connection with bone conduction. Recently the subject has been given much more attention by some men like Schoen and Goldberger, Claus and others. The ears of the patient are connected with those of the examiner by means of a double phonendoscope, and the fork then placed on the patient's head, later on that of the examiner. It is claimed that thus an objective control of the patient's subjective statements is had.

Diagonal or Crossed Resonance or Perception: When the fork is placed on the median line of the skull it divides it into two equal portions and the sound is not lateralized, but if the fork is set external to the median line, the skull is divided into two unequal vibrating masses, the smaller of which is adjacent to the central stem of the

fork, and the larger on the opposite side of the head. Just as with the Weber test, so with the resonance test, some individuals may have great difficulty in appreciating the matter of lateralization, and secondly, the sound is often lateralized to the side contrary to the one that would be expected from the nature of the lesion.

Resonators and Their Possible Use in Functional Testing of the Ears: For a number of years the writer has been experimenting with the sonators, especially those of the Schaefer set in an endeavor to see whether their use would be of value in functional testing of hearing in conjunction with the tuning fork findings. Resonance or sympathetic vibrations, which is the reinforcement or intensification of sounds due to the union of direct and reflected waves, depends upon the principle that a number of slight impulses, properly applied, will finally create a considerable momentum as seen in the well known fact of giving impulse to a swing or pendulum at the proper phase of the oscillation. It is easy with the resonator to test the actual duration of vibration of the various forks. In determining the presence of complete deafness for certain tones, resonators are of great aid, for if a fork, especially one whose pitch lies in the so-called "speech area," is not heard at all when amplified by the resonator, hearing for that tone can be said to be absent for all practical purpose. It may be possible to determine the pitch of a tinnitus aurium from the patient's own observation when the resonator is attuned to the various sound in the surrounding air. Resonators may be used for certain definite tones, such as A (108 d. v.), c1 (256), c3 (1024), and if the fork is struck with definite intensity and held before the proper resonator, you can measure the distance heard. This method gives more even distribution of sound than by holding the fork before the ear, for it may turn in the fingers or touch the auricle; with the resonator, there is also less interference with the sound waves from the fork. Resonators show their greatest efficiency in the middle octaves, being much less powerful in the lowest and in the highest pitches. Decrease in the efficiency of the resonator with a rising pitch is due to the unfavorable phase relation of the air pressure produced by the tuning fork with that caused by the resonator.

In association with Dr. John P. Minton the writer conducted some studies on the reinforcement of sound by means of the Schaefer resonators. Our conclusions were that the sound intensity emplification with the resonators depends on the manner in which they are used. The maximum value of this amplification as observed in a closed room, first by the examiner in 273, secondly by the patient, 44, and thirdly, as it appears in an open, unconfined space, over 10,000. The

efficiency of the Schaefer apparatus in common with other types of resonators decreases rapidly in approaching the higher pitched tones. The same is true of lower ones except when tested in the open, unconfined space. Work with resonators indicates that their use may have some significance, but to really decide their actual clinical value, if any, in otology as an aid to diagnosis, prognosis, etc., will require further extensive investigation.

The Records of Functional Tests: There has always been great difficulty in devising a method of properly recording tests so that the information therein contained would be readily accessible to and understood by all readers, as well as give a reliable index of findings for the examiner himself. With some slight modification I have employed the acoumetric formula proposed and adopted at the eighth otologic congress in Buda Pesth in 1909, and have combined it with the diagram long used in the Vienna University Clinic. With either of these methods it is easy, in a few minutes, to record the tests and have all the information available in a small, compact space. Many otologists may develop for themselves schemes which seem adequate, but it is highly desirable that some universal method be devised which would be understood by readers in all parts of the world.

Audiometers and audiometric measurements will be discussed by one who has had wide experience with them, Dr. E. P. Fowler, so I will therefore merely quote a few words from the recent report of the Committee on Standardization of Tuning Forks and Hearing Tests of the American Academy of Ophthalmology and Otolaryngology of which I have the honor of being chairman since it was appointed in 1921. "Considerable work has been done with audiometers of various types during the past few years. While these apparatuses are no doubt of great value in furnishing audiograms which give a graphic idea of the state of hearing of the individual, still we feel that they are merely adjuncts to, and cannot as yet replace the voice, tuning forks, whistles and monochord in functional testing. In the first place, the audiometers are quite expensive; then, owing to the various types, no definite standardization has as yet been made. We believe further work with them should be carried on so that within a few years there may be more uniformity in the apparatus offered. With most audiometers used at present, it is not possible to properly test bone conduction, but with the tuning forks this can very easily be done. Neither the lowest tones, from 64 down to 16 d. v., nor the highert ones, 9,000 to 22,000 d. v., can be tested with the office type of apparatus, but only by means of the very large, very expensive model suitable for research work. (I am here referring to the types developed by the Bell Telephone Laboratories.)

The hearing of the human voice (unaccentuated whisper or conversation) is the most important criterion of aural efficiency, even though it cannot well be standardized, but from a practical standpoint sufficient data may be obtained with the voice, without any apparatus if simple principles are observed regarding the closing of the eyes, occlusion of the opposite ear, etc. With the audiometers so far as we are aware, voice tests cannot be made except with the phonograph attachment. As previously mentioned, by using the Fletcher "constant" of damping decrement we can employ forks and get curves showing the loss of hearing in sensation units just as with the audiometer. This method, however, is more tedious when many pitches are being tested. On the other hand, this method has its advantages, the instrumentarium being much less expensive, etc., as previously mentioned. Meanwhile we suggest that the fundamental tests with forks be carefully and systematically done with the minimum armamentarium, which we have recommended."

Differential Diagnosis of Middle and Inner Ear Disease: One of the most important purposes of the functional testing of the hearing is, as stated at the outset, to determine the localization of the impairment of hearing. Naturally the history of the case, the pitch of the tinnitus (if one is present), the occupation of the individual (working in a noisy place, such as a boiler factory, often produced change in the auditory nerve), the otoscopic examination, inspection of the nasopharynx, the nose and the pharynx all are essential factors. The differential diagnosis is along the following lines: With middle ear involvement the low pitched sounds are heard less than the high ones; that is, the lower limit is elevated. In a nerve lesion the high pitched sounds are heard less than the low ones, and the hearing for tuning forks from c2 (512 d. v.) upward is much diminished, *i.e.*, the upper tone limit is lowered; but if there is marked involvement, both high and low pitches are affected. With the modified Galton whistles and with the monochord, the highest tones in middle ear trouble are usually normal, but in the inner ear disease they are markedly reduced. The Weber test is usually lateralized to the worse ear in conduction impairment, and to the better one in inner ear disease, but there are a number of notable exceptions. With the Schwabach test the bone conduction is usually found lengthened in middle ear disease and usually shortened in inner ear disease; rarely may it be normal in the latter condition. With the Rinné test we usually have a negative reaction in the middle ear disease and a positive one with the inner ear affection. N.B.—The lower the pitch of the forks used, the more likely is the Rinné to be negative. The appearance of the drum membrane in middle ear disease often shows cloudiness, loss of

luster, retraction, thickening or perforation. N.B.—In otosclerosis the membrane tympani is often entirely normal or shows a pink color over the region of the promontory. With inner ear disease the drum membrane is often normal, but may of course show changes if there has been any middle ear disease at some time. After catheterization of the Eustachian tube the hearing is often improved (at least temporarily) in middle ear disease, but it is not made better when there is inner ear disease present. The outstanding features are, that the typical conduction apparatus impairment usually shows a diminution in hearing for the low tones, lengthened bone conduction and a negative Rinné; whereas in inner ear disease we are more likely to have shortened bone conduction, a positive Rinné and diminished hearing for the high tones.

RECAPITULATION OF PRINCIPAL HEARING TESTS.

1. Observation of the patient.
   Loudness of voice (usually loud in severe inner ear disease: usually low in marked middle ear disease).
   Close attention and evident lip-reading on part of patient, with often appearance of anxiety in effort to hear.
2. Otoscopic examination:
   Inspection of:
   1. Auricle.
   2. External auditory meatus.
   3. Tympanic membrane.
   4. Mucosa of tympanic cavity if perforation of drum membrane is present.
   5. Mastoid region.
3. Nasal, nasopharyngeal and pharyngeal examination.
4. Testing with speech.
   With patient's eyes closed or averted and opposite ear closed, use:
   a. Unaccented conversation or whisper employing high and low pitched numbers or words and combinations of high and low pitched sounds.
      Designate in feet or meters or subdivisions thereof the distances heard, or state if ad concham, or not at all. If unaccentuated whisper is heard 1 or 2 meters need not use conversation.
5. Inflation with Politzer bag or by catheter, and again test hearing with speech (whisper or conversation).
6. Tuning fork tests.
   Range of hearing;
   Lower limits from C2 (16 d. v.) upwards—*i.e.*, C1, C, c, c1, c2, c3, c4, c5 (4096 d. v.).

Opper limits: *a.* Higher forks, c4 (2048 d. v.) and c5 (4096 d v.); *b.* Galton whistle, preferably Edelmann-Galton whistle or Schaefer-Galton; *c.* monochord.

1. Weber test for lateralization. Fork placed on median line of vertes, forehead or root of nose.
   a. Normally heard in vertex ("in the head").
   b. Usually lateralized in worse hearing ear in conduction apparatus impairment. If both ears have middle ear affection sound goes to worse of the two ears.
   c. Usually lateralized in the better ear if disease of perception apparatus is present in other ear. If both ears have perception impairment, sound usually lateralized in the better ear.
2. Schwabach test for duration of bone conduction in the individual as compared with the normal, usual living control, or comparing with average hearing for the particular fork employed. Fork usually placed on median line of vertex or forehead; it may be sent on mastoids. Note whether bone conduction is normal, lengthened or shortened, a slight or diminution being of no significance. The age of the patient, the thickness of the hair or bones, the manner of application of the fork, firmness of contact of fork, etc., may give variation in length of bone conduction.
   A definite lengthening of bone conduction means impairment of conduction apparatus (adhesions, fixation of stapes, etc.).
   A definite shortening of bone conduction means involvement of the perception mechanism (inner ear or auditory nerve).
   A decided change in bone conduction is in many ways the key to diagnosis and prognosis in ear disease.
3. Rinné test, for comparison of air with bone conduction in the same individual. The stem of the fork is placed on the mastoid (avoiding contact with auricle), and when no longer heard the prongs are held close to (without touching auricle or vibrisse) and parallel with the ear, and the duration of hearing by air noted.
   If a negative Rinné is suspected, *e.g.*, if the Schwabach was found lengthened, test air conduction first and then bone conduction.
   Normally the Rinné is positive (air conduction longer than bone). There are about eight varieties of Rinné (two forms of the positive, three varieties of the negative, two of the indefinite or plus-minus type and the so-called "infinite" negative, where there is no hearing via air, with slight hearing via bone).

4. Gellé test, for determining mobility of the foot plate of the stapes. Compressing the air in the external auditory meatus gives diminution of hearing in normal cases by pushing stapes into ovel window; where fiixation of stapedial foot plate is present no change in hearing occurs with increase in air pressure.
5. Stenger test, for unmasking simulation of total unilateral deafness. Two forks of exactly the same pitch used; patient unaware that more than one fork is sounding. The fork nearer one ear drowns out sound of fork at other ear.
6. Audiometers are being used considerably in testing and with them we get accurate measurements of hearing in sensation units. Audiograms obtained give a graphic idea of the state of hearing which may bepreserved for future reference and comparison. With properly calibrated forks provided with the "constant" of damping or decrement one is able to obtain a similar curve, but if many pitched are to be tested the process is somewhat more tedious than with the audiometer.
7. Resonators and other appliances should also be employed as previously mentioned in the Methods of Functional Testing of Hearing.

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THE MANAGEMENT OF INTERNAL EAR INFECTIONS. REPORT OF SIX CASES.

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The circulating fluids of the internal ear having a direct connection with the spinal fluid, it would seem that all labyrinthine infections must invariably develop a diffuse meningitis. This does not occur. Infections, apparently of the same type, with similar symptoms, will recover in one instance and be followed by a fatal termination in another. The most plausible explanation of the recoveries is that nature, by means of plastic exudates or adhesions, is enabled to successfully localize or wall off the infection from the other intracranial structures. The fact that this localization does occur in some cases suggests the lines along which treatment should be conducted.

The labyrinth, while exceedingly small, has both a direct and an indirect connection with the spinal fluid; the direct is through the vestibule, perilymph, aqueductus cochlea and spinal fluid; the indirect is through the endolymph, aqueductus vestibule and subarachnoid space. Although it may be impossible to differentiate them, there seem to be two different types of infection—direct and indirect. The first is circulatory in nature, the infection being introduced directly into the labyrinth and the spinal fluid by the blood or lymphatics, with little if any time for nature to form protecting exudates or adhesions; the infection is overwhelming, of short duration and quickly fatal. The indirect type is due to an extension to the internal ear of an old middle ear or mastoid infection, with time for protecting adhesions to form, and with symptoms which warn of a labyrinthian involvement and offers a fair chance of an operation checking the infection. At the present time a diffuse meningitis is practically always fatal, a successful result will therefore be possible only in a certain per cent of the cases.

The recognized symptoms of labyrinthitis are marked vertigo, nystagmus, nausea, vomiting and dizziness. These may also occur from an infection in the cells around the labyrinthine capsule, and it is therefore imperative that this type be differentiated from a true labyrinthine infection. It may be necessary to open the labyrinth when there is an intracapsular infection; it is absolutely contraindicated in the other. Marked headache and temperature usually mean an extension of the infection to the meninges.

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The Vaue of a Spinal Fluid Analysis: It is unfortunate that some of the leading authorities condemn this procedure. The theoretical argument is advanced that the removal of the spinal fluid in the presence of an active labyrinthitis will allow the infection to be drawn into the spinal canal. The writer has carefully investigated this through a number of different colleagues, and has failed to find a single instance where it has occurred. The information to be gained is of the utmost importance, and in many instances the question of operation depends entirely upon this analysis. An increase in the cell count of the spinal fluid occurs before the physical signs of a meningitis; a repetition of the analysis furnishes an accurate idea of the progress of the infection. From the writer's viewpoint, therefore, it would be impossible to intelligently advise for or against operation in a given number of cases without the aid of this analysis. Each labyrinth case presents an individual problem, with its treatment depending on the results of a careful physical and functional examination, together with the findings in the spinal fluid.

Operation is by no means indicated in all cases. Care must be taken that operation does not induce rather than prevent a meningitis. The infections may be divided into four groups: Perilabyrinthitis, circumscribed labyrinthitis, diffuse latent labyrinthitis, diffuse manifest labyrinthitis.

Perilabyrinthitis: This is not a true labyrinth infection. The symptoms are due to the inflammation of the bony tissues around the labyrinthine capsule, especially where there is an extensive pneumatic cellular development in the region of the posterior and horizontal semicircular canals. A functional examination will show an active labyrinth. Any operation necessitating the opening of the internal ear is absolutely contraindicated. A complete simple or radical operation will usually result in prompt recovery.

Circumscribed Labyrinthitis: Here there is a localized necrosis of the internal ear, usually in the form of a fistula in one of the semicircular canals; there may or may not be a positive fistula test present; the labyrinth is active. A spinal fluid analysis should be made and, if normal, a radical mastoid operation should be performed and the labyrinth capsule inspected. If the area of necrosis is small, and apparently walled off, nothing more is indicated. Many cases will recover without further complications or operation. If necessary, a labyrinth operation may be done at any subsequent time. The old idea that a radical operation should never be performed in a case of labyrinthitis without doing a labyrinth operation has changed. The decision rests entirely upon the findings in the individual case. In the

absence of a positive fistula test, the diagnosis is frequentlly not made, the fistulous opening being discovered at the time of the radical operation. In this type the essential idea is that functional activity is still present, and a labyrinth operation should be deferred.

Diffuse Latent Labyrinthitis: This means total destruction of the function of the internal ear without symptoms of an active labyrinthitis. There may or may not be a purulent labyrinthitis present. The spinal fluid analysis should be normal. In the event of a healed otitis media purulenta chronica, no surgical intervention is necessary. In the presence of active chronic otitis and mastoiditis, a radical operation should be performed. If there is no visible evidence of a labyrinthine necrosis, this alone will suffice. If evidence of necrosis of the internal ear is present, conservative drainage of the labyrinth by means of a Hinsberg labyrinth operation is best, care being taken not to open through the cochlea into the spinal fluid. In this type the infection has been localized or successfully walled off from the meninges, and it is imperative that nothing be done to interfere with the protection.

Diffuse Manifest Labyrinthitis: The real difficulties are encountered here. The marked nystagmus, vertigo, nausea and dizziness leave no doubt as to the diagnosis in this type. Some of them will recover without operation; others will go on to a diffuse meningitis. At times, the decision is most difficult. Analysis of the spinal fluid is the best guide. Where it is normal, a labyrinth operation may be delayed. With an increasing cell count in the spinal fluid, drainage of the labyrinth may check the infection. A Hinsberg operation should be done if there are no bacteria in the fluid. When there are bacteria in the fluid, drainage through the posterior fossa with a dural incision may be tried.

It is evident from the foregoing classification that the indications are fairly definite in the first three groups. There may be a difference of opinion in regard to the fourth. The whole effort is centered upon the attempt to confine the infection to the labyrinth or, if possible, check its intracranial extension. A brain abscess, sinus thrombosis, diffuse meningitis or encephalitis will always be responsible for a certain number of fatalities. Possibly the future holds a successful method of treating a diffuse meningitis; if so, the problem will be very much simplified.

CONCLUSIONS.

An increase in the cell count of the spinal fluid will occur before the manifest symptoms of a meningitis are present.

A labyrinth operation is absolutely contraindicated in cases of perilabyrinthitis.

A labyrinth operation is also contraindicated when the functional activity is still present.

In the diffuse latent type, successful localization has been established and opening the dura or opening through the cochlea into the spinal fluid is contraindicated. In the event that a labyrinth operation is necessary, the Hinsberg method is the best.

In the manifest type, the writer does not believe the dura should be opened or the Neumann operation performed when there is an absence of free bacteria in the spinal fluid.

CASE REPORTS.

Case 1. Perilabyrinthitis: Female, age 28 years. O. M. P. C. right ear since infancy, left ear normal. Last few months, recurring attacks of dizziness, nausea, vertigo, occasional vomiting. Horizontal nystagmus to the left, slight facial weakness right, labyrinth active, spinal fluid normal. Radical mastoid operation: middle ear and antrum filled with granulations, ossicles destroyed, with an extensive necrosis of bony tissue around posterior semicircular canal and along vertical course of the facial nerve. No visible labyrinthine necrosis, small area of cellular destruction left in this region for fear of facial paralysis. Radical cavity dermatized, but small fistula remained in the region of the posterior semicircular canal with very foul-smelling discharge. Dizziness and nystagmus continued, labyrinth active. Ten weeks later, secondary operation, which failed to stop the discharge and symptoms. Eight weeks later, third operation. Patient advised of probable facial paralysis, bony tissues very thoroughly removed behind and below the posterior semicircular canal and around the facial nerve. Facial twitching occurred four times during operation; no facial paralysis. This was followed by prompt recovery. Dry ear, with cessation of all symptoms.

Case 2. Diffuse Latent Labyrinthitis: Female, age 34 years. O. M. P. C. right ear since infancy. Large polyp filling the canal to the external auditory meatus, *labyrinth reported active,* left ear normal. Radical operation showed dense sclerosed bone, antrum and middle ear filled with pus and granulations, necrosis of ossicles. The polyp was fibrous and very firmly attached to the promontory, with necrosis involving the cochlea. Complete radical, with the removal of the polyp to its base. No evidence of pus in the cochlea, granulations apparently healthy, a labyrinth operation was not performed. After the first dressing, the *labyrinth was found dead,* with a negative response to both hot and cold caloric tests, Weber left. Necrotic area

was apparently filled with healthy granulations. Function had undoubtedly been destroyed for some time, with an erroneous report of an active labyrinth before first operation. Spinal fluid normal, no further operation, recovery uneventful.

Case 3. Diffuse Latent Labyrinthitis: Male, age 24 years, O. M. P. C. left ear, several years' duration, right ear normal. Large polypy completely filling the external auditory canal, labyrinth dead, Weber referred to the normal ear, spinal fluid negative. Radical operation disclosed very thick sclerotic cortex, antrum and middle ear packed with dense granulation tissues, posterior portion of horizontal semicircular canal destroyed, necrotic area apparently filled with healthy granulations. Complete radical operation performed, but no labyrinth operation. No evidence of facial paralysis, complete recovery.

Case 4. Diffuse Manifest Labyrinthitis and Meningitis: Male, age 22 years. O. M. P. C. left ear, fifteen years' duration. Radical mastoid six weeks previous. History of the removal of granulation tissue from radical cavity by an assistant in the clinic on Friday afternoon, two days later, complained of headache, dizziness and vomiting. Examination at 2 p. m. on the third day. Patient entirely conscious, normal temperature, neck not stiff, slight Kernig. Very dizzy. Complained of headache. Slight horizontal nystagmus to the right. Spinal puncture ordered, fluid clear, under pressure, with normal cell count. Immediate microscopic report before culture showed *free streptococci in the fluid.* Within twenty-four hours, all symptoms of diffuse meningitis; unconscious, neck very rigid, marked Kernig, temperature 104.6.° Condition unchanged end of sixth day. Second operation. Radical cavity thoroughly exenterated, promontory removed, opening made through cochlea to the spinal fluid, which flowed very freely and was apparently clear to the eye. Patient began to improve. Temperature came down to 100.6.° Regained consciousness. Stiffness of neck disappeared. Slight Kernig remained. Was able to take nourishment. Symptoms of diffuse meningitis slowly recurred. Five subsequent spinal fluids all showed a high cell count with streptococci present. Patient died of a diffuse meningitis.

Case 5. Diffuse Manifest Labyrinthitis and Encephalitis: Male, age 43 years. O. M. P. C. right ear, forty years' duration. Patient first seen at home, very dizzy, nauseated, occasional vomiting. Temperature 101.5,° with spontaneous vertical rotatory and horizontal nystagmus more marked to the left. Nystagmus unchanged in either erect or prone position, also unchanged following caloric test.

Slight intermittent delirium, labyrinth dead right ear, left ear normal. Immediate operation. Spinal fluid taken on table before operation showed 750 cells, no bacteria. Mastoid, dense sclerosed bone. Radical operation showed destruction of posterior portion of horizontal semicircular canal. An opening was made through the hard angle into the vestibule, promontory removed, together with the bridge between the oval and round windows. The following day patient was much improved, temperature 100,° mentally clear. Second day spinal fluid showed 1,250 cells, no bacteria. Third day, temperature 100,° but mind hazy, complained of severe headache. Probable diagnosis of brain abscess made. Exposure and exploration of cerebellum was negative. Exploration of cerebrum showed same result. Condition gradually grew worse, patient died on the fifth day, temperature 100,° cell count spinal fluid 500, no bacteria. Post-mortem refused. Death was not due to a diffuse meningitis; there was, however, an undoubted intracranial extension of the labyrinth infection, probably an encephalitis. It was interesting to note the fact that the spontaneous nystagmus in all directions was absolutely unchanged following hot and cold caloric tests, in both erect and prone positions.

Case 6. Diffuse Manifest Labyrinthitis and Meningitis: Patient was operated upon by the writer immediately after first examination. Previous history, as accurately as could be obtained, was as follows: Male, age 26 years, O. M. P. C. right ear, eleven years, left ear normal. For several months intermittent headaches, dizziness, nausea, occasional vomiting. In the latter part of July was brought to the hospital in an ambulance. Marked dizziness, nausea, headache, vomiting, nystagmus left, temperature 101° to 102.° After a few days, symptoms subsided. Operation refused, patient left hospital without permission. Two weeks later was returned to the hospital in an ambulance, with recurrence of all symptoms. Neck slightly stiff, partial Kernig, temperature 102,° spinal fluid slightly turbid, cell count 2,080, no bacteria. Wassermann on fluid negative. White blood count 16,000, polys. 65 per cent, operation still refused. Eight days later, spinal fluid cloudy, cell count 18,100, white blood count 26,000, polys. 78 per cent. One week later, patient was first examined by the writer. Complete deafness right ear, with dead labyrinth, tuning forks referred to normal ear, practically no response to caloric. Middle ear filled with foul-smelling granulations, temperature 99.6.° Partial facial paralysis right side. Active labyrinth symptoms subsided. Complete radical operation showed extensive labyrinthine necrosis, there was destruction of the upper and posterior portions of the horizontal semicircular canal, with a fistulous opening

leading through the hard angle into the vestibule. Promontory was destroyed, but the thin bony canal containing the facial nerve as it passed between the oval window and the horizontal semicircular canal was intact. Granulations were thoroughly removed, vestibule was cleaned out, after which a small sponge could be passed through the hard angle above under the facial nerve and out through the vestibule below. The bridge between the oval and round windows was removed, the dura exposed in the middle fossa and found normal, no exposure was made in the posterior fossa. Facial paralysis entirely recovered in about eight weeks. Particular care was taken not to open through the cochlea into the spinal fluid, patient made a complete recovery. There was undoubtedly a meningitis present, as shown by the cloudy spinal fluid and high cell count. Recovery was probably due to the fact that there was no bacteria in the spinal fluid.

COMMENT.

In the first case, despite active signs of labyrinthitis, a normal spinal fluid and an active labyrinth contraindicated a labyrinth operation. Thorough removal of the surrounding infection resulted in a prompt recovery.

In the second and third cases, both labyrinths were dead, the infection was successfully walled off from the meninges, with normal spinal fluids. In view of the fact that the necrotic areas involving the internal ear were apparently filled with healthy granulations, labyrinth operations were not performed, and they promptly recovered.

In the fourth case, there was a diffuse meningitis from the beginning. It is interesting to note the marked improvement following the drainage of the spinal fluid through the cochlea, also the fact that free streptococci were demonstrable in the clear spinal fluid with a normal cell count.

In the fifth case, the spontaneous rotatory vertical and horizontal nystagmus, unchanged in erect or prone position, following both hot and cold caloric tests, indicated brain lesion from the beginning, probably an encephalitis.

In the sixth case, recovery followed a labyrinth operation, despite the fact that there was a cloudy spinal fluid with 18,100 cells present.

123 East 53rd Street.

PROCEDURE SUGGESTED FOR THE DIAGNOSIS OF SINUS THROMBOSIS.*

DR. REUBEN OTTENBERG, New York City.

Last year on the medical service at Mt. Sinai Hospital we had as a patient an old man with a pyemia. He gave a history of having had an otitis media some weeks before, but it had entirely subsided at the time of his admission. The problem arose as to whether a sinus thrombosis was at the basis of the pyemia. Thinking over some possible way of solving this problem without operation led me to subsequently develop the procedure which I am about to suggest to you. In the particular case of the old man I did not have the opportunity to try the procedure because by the time I had studied the anatomy and experimented on obtaining blood from the internal jugular vein of normal persons the old man had been cured by the ordinary surgical procedure of opening and draining his multiple abscesses. Incidentally, in this instance Dr. Friesner's judgment, based on pure clinical evidence that it was not necessary to operate on the lateral sinus, turned out to be correct.

The procedure is simply this: One takes a blood culture from the internal jugular vein in both sides of the neck. One plates out accurately measured quantities of the blood so as to be able to count the exact number of colonies per cubic centimeter of blood from each internal jugular vein. A striking difference in the number of bacteria in the two sides should be construed as evidence that bacteria are being fed into the blood of one side or the other from a thrombus.

Naturally I first looked up the literature on the subject to determine whether the procedure had ever been tried before, and in a rather thorough study in the course of which I covered over fifty articles published since 1907, I have not found any reference to this having been done. The nearest thing to it was done by Dr. Ernst Leutert, of Giessen, whose fundamental studies opened up the whole subject. In one case of sinus thrombosis at operation at the time when the internal jugular vein was exposed in the neck, he took a culture from it and simultaneously took a culture from the patient's arm vein. He found a very large number of bacteria in the blood from the internal jugular vein and none in that from the arm vein and, therefore, the next day when this finding was obtained he reopened the wound and

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ligated the internal jugular vein. Libman in his classical studies in 1912 discusses this procedure but does not seem to have tried it. Kopetzky, in 1926, again refers to the possibility, but in general the procedure suggested by Leurtert of culture from one internal jugular vein and from a peripheral arm vein at a time of a preliminary operation and then waiting for a report before tying off the internal jugular vein appears to have fallen into neglect.

The procedure of obtaining blood without operation from the two internal jugular veins does not seem to have been tried at all.

The procedure for obtaining blood for culture from the internal jugular vein has turned out to be not extremely difficult and after some experiments on the cadaver and on normal human being I have developed the following technique: the patient must be inverted on an operating table in the Trendelenburg position to an angle of 45.° This is essential and the only two cases among nineteen on whom I have tried to obtain blood from the internal jugular vein in which I failed to obtain blood were cases which could not be inverted. The patient is instructed to hold his breath and press. The internal jugular vein can then be felt as an elastic swelling at about the level of the thyroid cartilage. At this level it lies more superficial than at any other part of its course, being covered by the anterior part of the sternomastoid muscle. The needle is inserted over the muscle at about the level of the hyoid bone and is pushed downward and inward through the muscle until it enters the vein. The needle is a fine one (19 gauge, 1¾ inches long). I believe that if one knows one's anatomy sufficiently well to keep away from the trachea and the esophagus that this procedure is entirely safe. The carotid artery lies decidedly deeper than the internal jugular vein and anyone who has tried to obtain blood from arteries knows that it is very much more difficult to put a needle into an artery than into a vein. However, if one did pierce the artery one would do no harm, as needles of this size are regularly used to puncture arteries to obtain blood for blood studies. The large nerves lie so much deeper that one hardly could pierce them and here also the smooth, round surface of the nerve would probably make it slip away from the tip of the needle, if the needle did encounter the nerve. Even if this did occur it seems unlikely that so small a puncture could give any trouble.

I have now punctured one or both internal jugular veins on seventeen persons. In seven of these it was done for blood cultures and these cases will be discussed below. In ten of them blood was obtained for some other clinical procedure, such as Wassermann reaction or blood chemistry. Two of the cases were infants, approxi-

mately 1 year old, in whom repeated attempts to obtain blood for diagnostic purposes from some other vein had failed, in which the internal jugular vein puncture was successful.

The blood is plated in glucose agar plates and 2 c.c. of ascitic fluid added to each plate. It is important: *1.* that the ascitic fluid be warmed to body temperature before it is added to the agar and blood; *2.* that the blood should be plated very promptly so that no beginning coagulation occurs; and, *3.* that mixing be very thorough by pouring from tube to tube so that the count on the two sides will be strictly comparable. The colony counts are best expressed in cubic centimeters of blood. In addition to the plates it is desirable in cases where there has been no previous positive blood culture to inoculate blood in glucose serum flasks as occasionally one obtains a positive finding in this optimal medium when agar plates are sterile.

Of the seven cases in which blood cultures were taken from both internal jugular veins, three gave negative results on both sides. There were:

Case 1: A case of hemorrhagic otitis with subsequent uncomplicated recovery.

Case 2: A case of suspected sinus thrombosis in which the subsequent recovery without operation disproved this diagnosis.

Case 3: A case of suspected sinus thrombosis in which in spite of the negative blood culture an operation was done and an occluding thrombus was found.

Of the four positive cultures one was a control observation on a case of bacterial endocarditis in order to determine the question of the accuracy of the method. In this case the average number of colonies per cubic centimeter of blood from the right internal jugular vein was fourteen and from the left was 10½. The other three cases of positive blood culture were cases of sinus thrombosis.

Name	Diagnosis	Colonies per c.c. Right	Left	Operative findings
1. Stearn	Sinus Thrombosis	278 Hemolytic streptococci	9	Left sided thrombus (occlusive)
2. Mina	Sinus Thrombosis	25 Hemolytic streptococci	8	Left sided thrombus (not seen but prompt recovery after ligation)
3. Greenfield	Sinus Thrombosis	2	1	Right sided thrombus (occlusive)
(Control Case) Sugarman	Bacterial Endocarditis	14 Streptococcus viridans	10	

The first case is the one which Dr. Druss will present to you in full in a few minutes and to which, therefore, I will only briefly refer. The interesting point is that it was a case in which there was considerable doubt as to wether sinus thrombosis existed at all and, if so,

as to which side should be operated on. The double jugular blood culture showed a huge preponderance of colonies in the blood from the right vein, 278 colonies on this side as compared with nine on the other. Accordingly, Dr. Maybaum exposed the right lateral sinus first and found it normal. He then exposed the left and found a very extensive occlusive thrombus. I was at first chagrined at finding the large number of bacteria in the blood stream from the normal side, but on reconsidering the technic in every detail it was certain that no mistake had been made. It was, therefore, necessary to seek for an explanation of the surprising fact, and two factors presented themselves, either or both of which may have been involved. *1.* The lower end of the thrombus may have already sterilized itself so that bacteria were only being fed into the blood stream from the upper end. Dr. Libman tells me that he has seen such cases. *2.* The bacteria may have been growing in approximately equal numbers in both ends of the clot, but due to the fact that the clot was occlusive there was practically no circulation in the upper end of the internal jugular vein, which would under these circumstances be a blind *cul-de-sac*, receiving no important tributaries, while the blood in the lateral sinus would be in active circulation, due to the considerable volume of blood received from the petrosal sinuses and this blood would have practically no other means of exit from the cranium than by reversing its current and going through the Torcular Herophili and through the opposite internal jugular vein.

These considerations would lead one to expect that in a strictly mural and nonocclusive thrombus one would find a diametrically opposite result, namely, a large number of colonies in blood from the diseased side. Only experience will tell whether this will be the case. If it is, the usefulness of the procedure for determining in which side the thrombus lies will be limited. However, the great majority of thrombi found at operation are occlusive and the mural thrombi are found chiefly in very early cases and a consideration of these facts may possibly enable one to evaluate the preponderance in one side or the other after all.

The second case was a private case, for which I am indebted to Dr. Lempert. In this patient 8½ colonies were found on one side and 25 on the other. The thrombus, which was incompletely occlusive, was found on the side with the smaller number of colonies, as in the first instance. Whether a difference of this magnitude is of significance I cannot tell with my present limited experience.

The third case, which, like the first, was on Dr. Friesner's service at Mt. Sinai Hospital, presents certain other remarkable features and

will very likely be presented by him or another one of his assistants at another time. In it a diphtheroid bacillus was isolated from the ear and was twice present in blood cultures before the double jugular blood culture was done. Here the diphtheroid bacillus was again found and in approximately equal but small numbers on both sides. At operation a completely occlusive thrombus was found in the right lateral sinus.

From the small amount of work done so far one cannot say whether the method will be of value in determining on which side the thrombus lies. Possibly with a larger experience it may be of some help in this direction. The only situation in which it seems so far that the method may be of value is one in which in the presence of a positive blood culture one is still in doubt as to whether the bacteria are coming from a sinus thrombosis or from some other possible source, such as for example ,a complicating pneumonia or endocarditis. Under these circumstances as in the first of the three cases here reported the finding of a very large difference between the two sides would be of positive value. The finding of approximately equal numbers of bacteria on the two sides would, on the other hand, be of no value in excluding sinus thrombosis.

15 West 89th Street.

JUGULAR BULB THROMBOSIS, MULTIPLE ABSCESSES. RECOVERY.*

DR. FLOYD C. MCDANIEL, New York City.

The patient, a boy, age 10 years, first consulted me on June 2. The general physical examination was negative. No history of any ear infection until a week previous, when he had suffered from an acute cold and had complained of slight deafness in right ear. For three days he suffered acute pain and an increased temperature.

When seen, his eardrum was reddened, thickened and bulging, and there was a slight sagging of the posterior canal wall. A myringotomy was done immediately and found a serosanguineous fluid under pressure.

The following day his temperature was 104,° there was a slight tenderness over the mastoid tip and a marked pulsation through the

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incision. I advised his removal to the New York Eye and Ear Infirmary, and the following reports were obtained:

X-ray of the right mastoid showed cloudy throughout, especially in the antrum, but no indication of breaking down. Sinus not shown.

A smear from the canal showed short chain of streptococci.

Blood count: Hemaglobin, 90 per cent; red cells, 4,500,000; White blood cells, 14,000; polynuclear, 75.5 per cent. Blood culture was negative at twenty hours. Urine negative.

The next day, June 4, his temperature was 106.4,° but he had had no chill. The mastoid was opened the same afternoon.

On removing the cortex it revealed hemorrhagic condition of the cells. There was free pus in antrum. One or two large type cells contained free pus under pressure. Sinus plate apparently healthy and no exposure of sinus. Dural plate healthy.

Patient stood operation well and the following day temperature was down to 101.6,° but rose to 105.8° about noon. Examination of the blood culture at the end of forty hours showed one colony of hemolytic streptococci.

Dr. Atkins was called in consultation and we decided upon an immediate jugular resection, and the following conditions were noted:

The jugular vein showed evidence of periphlebitis with involvement of lymphatic glands, but no clot was demonstrated. The lateral sinus plate was removed and the sinus opened; there was no clot. Free bleeding from above and very little below. Following the jugular resection, his temperature ranged from 101° to 104° for several days, and for the following forty-eight days from 100° to 105.° Then from July 21 to August 4, it ranged from 99° to 102.° The highest peak of his temperature was 106.4,° which was reached on several occasions. He had continuous temperature from about June 1 to Oct. 3.

Numerous blood cultures were made and remained positive for eight days following the jugular resection. Hemolytic streptococci was the organism found each time.

From onset of illness he complained continually of pain in the region of the right hip and thigh. X-ray examinations were made and aspirations of hip and thigh, which proved negative.

It was not until June 17 when first metastatic abscess manifested itself in the left elbow and the dorsal surface of the left wrist; incised and drained. Ten days later the abscess in right thigh and ankle, incised and drained. One in left shoulder was discovered a few days later. Physical examination showed a marked enlargement of the liver. Exploratory laporotomy was done, thinking that there

was possibly an abscess. Exploration revealed a marked enlargement of liver, which extended to the crest of the ilium, but aspiration in several points showed no evidence of abscess.

On July 10, the right hip joint was opened and an abscess found and drained. It had caused spontaneous dislocation of the right hip. Several more abscesses were opened, the last one being opened on August 22, and this was seventy-nine days following the jugular resection. In all, he had fourteen metastatic abscesses which were opened and drained, including an exploratory operation on the liver. The first abscess occurred seventeen days after operation and the last one seventy-nine days after jugular resection. The largest abscess was probably at the posterior part of right thigh, which contained 500 c.c.

The cultures from the hip and several abscesses revealed staphylococcus-aurus and colon bacilli. His blood picture in the beginning showed hemoglobin, 90 per cent—the lowest was 45 per cent. The highest white cell count was 14,000—the lowest, 8,000. The highest poly, count was 80 per cent—the lowest, 62 per cent.

The blood transfusions undoubtedly played a very important role in his recovery. He had, altogether, thirteen transfusions. In the beginning, while his blood cultures were positive, 250 c.c. were withdrawn and 500 c.c. of blood were given.

On August 4 he was moved to the Orthopedic Hospital, where an operation was done to reduce the dislocation of the hip, which has left him with an ankylosed hip joint. The involvement of the right knee has caused a partial stiffness. He has a 45° motion in this joint.

He was discharged from the Orthopedic Hospital on Oct. 3, has gained weight, all the old incisions have healed, and with the use of a cane he is able to be up and around and apparently recovered.

Some of the interesting facts in this case:

1. He had a blood stream infection when he was admitted to the hospital.

2. The pain in the right hip was evidently a metastatic abscess in the beginning, which pre-existed the other abscesses.

3. The bulba thrombosis was probably caused by infection through the venous or lymphatic channels.

4. Abscesses developing in parts other than joints were usually accompanied by little or no pain.

152 West 58th Street.

BUCCO-ANTRAL FISTULA — CLOSURE BY PLASTIC PROCEDURE.*

DR. IRVING WILSON VOORHEES, New York City.

This gentleman was presented before the Section in January, 1925. By way of refreshing the memory I will give a brief resumé of the history as then stated.

During January, 1924, the patient consulted his dentist for severe pain in the right upper jaw. At that time there were many decayed, badly infected teeth and general evidence of prolonged neglect. There was a sinus extending from the right upper molar region into the antrum, through which bare bone was felt. The dentist removed considerable bone from the diseased area, curetted the antrum, and attempted plastic closure, which did not succeed. The antrum was irrigated daily with a boric acid solution for about one month, when a second attempt of the same kind and nature was made, likewise with no good result. Following this, a rubber denture or obturator was made to fit the sinus in order that food, fluid and bacteria should not enter from the mouth. A few weeks later a further plastic operation was undertaken, but the sinus persisted and the result was about *nil,* save that the opening was rather larger than it was preceding the first operation.

On Nov. 5, 1925, the patient came to my office seeking relief from the foul discharge, which had taken away his appetite and was interfering seriously with his general health. At this time the sinus was about 1 c.m. long and 4 m.m. wide. A small amount of pus was seen coming down. In the right middle meatus two or three small polypi were seen. X-ray examination showed marked opacity of the right antrum; therefore it was decided to do a radical Caldwell-Luc operation at once. This was carried out at the Manhattan Eye, Ear and Throat Hospital on Nov. 16, 1925, under general anesthesia. The antrum was almost filled with polypi and foul pus, and at the very bottom of the cavity I came upon a hard mass lying free, which I took to be an imperfect, unerupted molar, but which several dentists identified as an odontoma.

After making the usual counter-opening in the nasoantral wall, the gingival margin was sutured, but the edges were so poor in viability that no union took place and the resulting fistula was about three times as large as when I first saw the patient. Shortly after this the

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patient was brought before the Section, and some of the gentlemen who saw him will remember the new granulation tissue and the crusting seen both in the antrum and in the nose.

Two months later, in March, the patient again entered the Manhattan Hospital and a plastic procedure for closure was successfully carried out. The margin of the fistulous opening was carefully cut away in a circumferential manner, and a free flap was dissected up on the hard palate area as far internally as the midline. On the opposite side a large flap was dissected up from the cheek in order to get as much "slack" as possible, thus making the approximation of the wound margins easier and reducing tension to a minimum. It was not at all difficult to do this, and three mattress sutures were passed from the palatal to the buccal flap, so that all tension on the suture line would be done away with. The wound edges were then closed with No. 1 chromic gut.

Postoperative care consisted in irrigating the nose two or three times a day with normal saline, folowed by dropping mercurochrome, 2 per cent, into the nose and brushing it along the suture line.

This is a very simple method of closing large fistulae which persist after tooth extraction. Whenever there is any considerable necrosis of the gingival margin of the superior maxilla, a fistula is quite sure to form. It is a well-established practice among dental surgeons to pull a tooth, usually the second cuspid or first molar, and to drill an opening into the antrum through which the antrum is irrigated daily for a long time. This I regard as probably the worst operation in all surgery, for the reason that it often converts a relatively simple condition into a chronic and prolonged suppurative one. Acute disease of the antrum frequently gets well either spontaneously or through the aid of intranasal irrigation, and where it persists, an opening into the nasoantral wall suffices to bring about healing within two or three weeks. If the antrum disease is a chronic one with degeneration of the lining mucous membrane, formation of polypi, and necrosis of any of the antral walls, then the best operation is the Caldwell-Luc done under local anesthesia by nerve blocking. It is almost as easy as a submucous resection, in some respects even easier to do, and the results are usually excellent. The amount of bleeding is fairly negligible as a rule, and in most instances I do not even use a postnasal plug. The wound beneath the cheek is sutured in every instance with fine chromic gut and healing goes on with a minimum of treatment. The patient can be dismissed in from fourteen to twenty-one days and comes in only occasionally thereafter for inspection.

114 East 54th Street.

REPORT OF AN UNUSUAL CASE OF TUMOR OF THE CAROTID BODY.

Dr. J. Eastman Sheehan and Dr. Max Rabiner, New York City.

Tumors of the carotid body are not frequently met with and relatively few cases have been reported. The one here described was so far out of the field in which they are usually found as not to have been suggested in preoperative examination.

The patient, a woman, age 50 years, entered our service at the Post-Graduate Hospital, New York, Oct. 21, 1925. She showed a swelling over the lower left parotid area, which she said had started two years before, after the extraction of a tooth. She had been treated during those two years with the X-ray and external applications of ichthyol. She now complained of difficulty in breathing.

She was kept under observation until Nov. 7. Roentgen examination was of no assistance; Wassermann was negative; nothing abnormal was indicated by the chemical and qualitative blood tests.

The conditions on examination appeared to favor diagnosis of an aneurism of the transverse facial artery. Pulsation was marked and there was a distinct bruit. On tapping with an aspirating needle blood was withdrawn. The swelling was larger on some days than on others. Occlusion of Stenson's duct, or tumor of the parotid gland, were alternatively suggested.

Incision was made beginning in front of the lower part of the ear, carried in a curve following the angle of the jaw, and terminating half-way along the mandible. Careful dissection followed, under control of adrenalization. As the dissection proceeded a tumor was found, enmeshed in and adherent to the parotid gland and surrounding tissues. It was different in appearance from tumors of the parotid, and of a brick dust color. It was not an aneurism nor was there occlusion of Stenson's duct.

On further dissection with a view to liberating it from its bed, it was found leading downwards toward the neck. The part exposed was thereupon delivered and excised and sent to the laboratory for frozen section. The part measured 34x34x23 m.m. Report was returned that it was tumor of the carotid body.

The neck was anesthetized for further dissection and a new incision made, bisecting the original incision and over the great vessels of the

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neck. By careful dissection, which took considerable time, the tumor, which became smaller as it descended, was followed to the crotch of the carotid artery, where it was carefully freed and removed.

After reassembling the tissues and acquiring a dry field the incisions were closed, using the subcuticular stitch.

Several days after the operation a partial paralysis developed at the left angle of the mouth and on the left eyelid. This cleared up in about three weeks, following massage and galvanism.

Comment: There was nothing to indicate preoperatively that the carotid body was involved in the swelling. There is no report, so far as we are aware, of a pedicle so long and so attenuated. The symptoms, while not conclusive, were those of an aneurism.

No recurrence has been reported. When last seen the patient appeared to be in good health.

The Carotid Gland: The carotid gland was discovered by Halle in 1762, and a monograph was written by Schnitzer in 1763. Luschka, who studied it, believed it to be a "carotid corpuscle." Streda and Delvene held it to have been derived from the walls of the carotid artery.

Normally, it is oblong in shape, with the longer axis vertical, 5 to 6 m.m. in length and 2 to 3 m.m. in width. Usually single, it has been found divided into two and three lobes. It is connected to the upper portion of the primary parotid by a pedicle 2 to 3 m.m. long which is composed of fibrous tissue and is of vascular structure (Mayer's ligament). The pedicle is attached at the lower pole of the gland treated as having been derived from the sympathetic chromaf-branchings from the bifurcation of the carotid; from the opposite side three or four veins pass to the supralaryngeal, lingual and pharyngeal veins.

While the physiology is not definitely established, the gland is assumed to be connected with the trophic stimuli of bodily development. Monlon has shown that the glomus caroticum, the corresponding body in mammals, is an organ of hypertension in adults; in the human the carotid body tends to atrophy after puberty or full body development.

There is disagreement as to its derivation. Several hold it to have been derived from the fourth branchial cleft, others from the penthelium of the carotid arteries, still others, that since it belongs to the chromaffin system, whose cells can only be produced from sympathetic tissue, the origins mentioned must be rejected and the gland, where also the gland receives its arterial supply from four fin anlage and identified with the intercarotid plexus. The body

responds to the tests for the chemical reactions of chromaffin tissue. (Cells stain brown by potassium bichromate, grayish green by iron perchlorid, red, brown or black by osmic acid vapor.)

The carotid gland is one of five organs classified as paraganglia (in adult humans), the others being the tympanic (along Jacobson's nerve), the cardiac (where the left coronary turns round the left

Fig. 1

Fig. 2

Fig. 1. Before operation, showing the size and location of the swelling.
Fig. 2. Three weeks after operation; slight paralysis at left angle of the mouth, which cleared up after treatments.

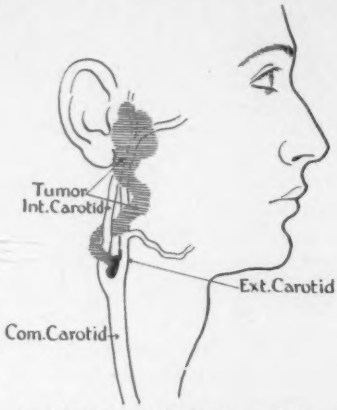

Diagram to show the location of the tumor and the course of the tumefied pedicle, over and under the arteries, down to the bifurcation of the carotid artery.

auricle), the aortic (where the inferior mesenteric is give off), and the coccygeal (tip of coccyx).

Diagnosis of Carotid Body Tumor: Ordinarily the tumor is located at the bifurcation of the carotid artery. It is movable laterally but not vertically. It is ovoid in shape, smooth and not lobulated. It occurs singly, only one bilateral case having been reported. Onset is silent, the tumor is of slow growth and there may be long stationary periods, followed by sudden increase in size. It is rather

firm and elastic, is neither tender nor painful, the main complaint being as to the deformity produced. In most cases it is found between the sternomastoid and larynx and the middle portion of the thyroid cartilage. The external swelling may be paralleled by bulging in the wall of the pharynx.

Much significance attaches to the pulsations; very distinct and readily explained by the situation in the vascular fork, they can be made to disappear by compression of the primary carotid. They are not accompanied by expansion. There is bruit and thrill and may be systolic murmur; similarity to symptoms of aneurism is usually noted on auscultation. Except when obscured as in the case here noted, bimanual palpation discloses the size and location; pressed backward, it comes in contact with the walls of the pharynx. In two instances a yellowish pigmentation was noted over the whole body, one light yellow, the other dark yellow.

The subjective symptoms noted in the literature are hoarseness, annoying cough, difficulty in phonation and swallowing, tinnitus, deafness and dental neuralgia, conjunctivitis, pressure on the laryngeal wall, dragging sensation in the neck and sometimes pain in the carotid area. In some cases also the patient is conscious of the pulsation.

Literature: Charles Scudder, of Boston, reported one case in 1903; Funke and McPath, one each in 1904; Keen and Funke collected 27 cases living and two at autopsy; Collison and MacKenty, 28 cases living, one at autopsy, and two discovered at autopsy. Of the 54 living cases, 31 were of males, 23 females. As to age incidence, three were before 20, and five after 60. In the decade beginning at 20 there were 10; from 30 to 40 there were 12; 14 from 40 to 50; 10 from 50 to 60.

In these 54 cases operation was followed by 12 deaths, to which are to be added four from recurrences and six in which death from recurrence was imminent; 32 recoveries.

Direct cause of death is given as pneumonia in four cases where the pneumogastric had been divided or resected; hemorrhage in three; cerebral anemia in two; and from acute edema of lungs, septicemia and infection and hemorrhage, one each.

Accidents to surviving patients included hemiplegia and aphasia, four; voice affliction with dysphagia, four; partial paralysis of face, four; tongue deviated to one side, five; constant cough and tracheal irritation, one.

In eleven of the cases cited the preoperative examination failed to indicate the presence of the tumor.

CONCLUSIONS.

Tumor of the carotid body is normally a hyperplasia of the gland; development of a malignant condition is rare, but a "potato tumor" of the neck may be a primary sarcoma or cancer of the carotid body.

The primary carotid and its branches are important aids to diagnosis, since the gland, which lies in the bifurcation, derives the pulsation, an invariable symptom, from them directly.

Paralysis of the vocal cords is associated with the bulging of the pharyngeal wall.

Sluggish mental condition, yellowish pigmentation and flushing about the head and neck may develop from the hyperplasia.

The position of the gland is not constant; it may be anterior or posterior to the bifurcation, and close to or considerably removed from the artery, according to the length of the pedicle.

While both objective and subjective symptoms are well defined, preoperative diagnosis cannot always be made with certainty.

Mortality in operation has been high. Involvement of the walls of the artery is a condition to be apprehended, and preliminary ligation before extirpation of the tumor is therefore to be considered.

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New York Post-Graduate Hospital.

OTITIC MENINGITIS RUNNING AN UNUSUALLY PROLONGED COURSE.*

Dr. S. Rosen, New York City.

A child, age 7 years, who nine weeks before admission to Mt. Sinai Hospital, took sick with measles. Three weeks later the child compained of right-sided headaches and pain in the right ear. At this time the mother noticed excessive tearing of the right eye, and called her physician, who noted a right peripheral facial palsy, and did a myringotomy. The ear discharged for about ten days. The head-

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ache, which became more occipital in location, persisted day and night, often keeping the patient awake at night. The facial palsy improved until it was very slight at the end of two weeks. As the facial paresis was improving, the child complained of a double vision for a day, and a weakness of the right external rectus was seen. For a week before admission to the hospital, child vomited two to three times daily, the temperature ranging between 99° and 102.° The child was admitted to Mt. Sinai Hospital on April 10. She was lying in bed on her right side, complaining of severe pain in the sub-occipital region. The positive findings were: right peripheral facial paresis, very marked rigidity of the neck, and bilateral Kernig.

The left ear was normal. The right drum was thick, the landmarks were not visible, and the hearing was good. There were no signs of postauricular disease. There was a bilateral optic neuritis, and a temperature of 105.° A lumbar puncture was done, 30 c.c. of turbid fluid with 2,840 cells, 80 per cent polys., was removed. Smear showed gram positive, diplococci. It was deemed advisable by the pediatrists to open the mastoid. This was done two days after admission, the mastoid containing pus. There was some necrosis of the cells. Culture of the pus from the mastoid showed an hemolytic streptococcus.

Because of the persistent meningeal signs, fever, headache and occasional vomiting, lumbar puncture was done almost every day—the fluid always containing from 12 to 1,800 cells, mostly polys., and gram positive diplococci on smear. The organism could not be grown.

As a last resort the cisterna magna was drained. Within forty-eight hours after this procedure the child became stuporous and ceased three days later.

The interesting feature in this case is the unusual length of time the child lived with a bacterial meningitis—five weeks after the first signs of meningitis, and eleven weeks after the first signs of Gradenigo's syndrome. There was no clinical evidence of mastoid involvement.

Mt. Sinai Hospital.

PRIMARY JUGULAR BULB THROMBOSIS PRESENTING CERTAIN UNUSUAL FEATURES.*

Dr. Joseph Druss, Brooklyn.

A. S., age 7 years, was admitted on Feb. 4, 1926, to Dr. Schick's service at Mt. Sinai Hospital, with a history that three weeks prior to admission he began to cough and had slight temperature. Three days later he complained of pain in both ears, which began to discharge at the end of four days. The temperature at that time was elevated to 104-105,° which was followed by an afebrile interval of two-three days, only to be followed by another rise to 105.° This type of temperature continued up to the time of admission. With each rise he complained of chilly sensation but there was no definite chill. About one week prior to admission the ears ceased to discharge and at about this time he began to experience vague pains around the umbilicus not associated with vomiting, and two days later he complained of pain in left hip, which persisted up to time of admission.

Past Illnesses: He had attacks of acute otitis media at the age of 2 and 3 years, and the usual infectious diseases, *i. e.*, measles, mumps and chickenpox. Had pneumonia at age of 5 years, and tonsillectomy following it.

Physical examination showed an undernourished, undeveloped boy of 7 years, with a waxy pallor resembling very closely the pallor seen in leukemia. He did not appear acutely ill. The cervical axillary and inguinal glands were somewhat enlarged but only the cervical glands on the left side were slightly tender. The liver and spleen were enlarged about 5-6 c.m. blow the costal margins, the edges being *hard, firm* and *sharp,* but not tender. The left hip joint was negative except for moderate pain on adduction of the leg.

There was a large central perforation in the right drum but no discharge. The left ear was also dry. There was evidence, however, of a recent acute otitis media in both ears. The remainder of the physical xamination was essentially negative.

For the first five days of the child's stay in the hospital, he ran a septic temperature, which was interesting, in that it reached 103-104° in the morning and declined to 100-101° later in the day.

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His blood count was of extreme interest and showed the following:

2/4—Hgb.	62%	2/6
RBC.	3,520,000	
Polys	48%	51%
Lymphs	25%	20%
Monos	5%	13%
Myelocytes	16%	13%
Myeloblasts	3%	2%
Myelogones	3%	2%

Urine was negative. Schick test was mildly positive. X-ray of the mastoids showed slight haziness over both mastoids but no definite destruction in either. Fundi were repeatedly negative. Blood culture was reported streptococcus hemolyticus, 60 colonies per c.c. His temperature remained septic in character and another blood culture taken four days later was again reported positive for streptococcus hemolyticus, the number of colonies increasing to 122 per c.c. In view of the streptococcemia with a definite increase in the number of colonies, the diagnosis of primary jugular bulb was strongly entertained and jugular ligation and sinus operation was considered. The question then arose as to which side was the site of pathology. With this in view a culture was taken from the blood of both internal jugular veins, as described by Dr. Ottenberg, and surprisingly enough it was found that the blood from the right internal jugular vein grew 275 colonies, while the left only 9 colonies. For this reason the right mastoid was first opened by Dr. Maybaum, and sinus wall exposed, but there was no disease in the mastoid except for slight sclerosis; the sinus wall appeared normal. A similar procedure was done on the left side and again the mastoid showed no pathology, but here the sinus wall appeared gray and was definitely thickened. Aspiration of the sinus failed to show the presence of blood. The internal jugular vein on that side was then ligated and the sinus operation continued. A large obliterating thrombus extending from the jugular bulb to about the level of the knee was removed. Bulb was curetted and drained.

Following the operation the child was transfused and reacted with a chill and temperature to 104.° A blood culture taken immediately after the operation and another forty-eight hours afterwards were both sterile. Except for the reaction following transfusion, the boy made an uneventful recovery; his temperature declined to normal and remained so throughout his stay in the hospital; his appetite became voracious, his weight increased considerably; the mastoid wounds and wound in neck healed satisfactorily, curiously enough; the liver and spleen gradually receded almost to their normal size. The blood

count became normal, the hemoglobin rising to 75 per cent. He was discharged April 7, *i. e.*, seven weeks after the operation, very much improved.

In summing up, I should like to emphasize the points of interest revealed in this case, namely:

1. The unusual blood picture of 16 per myelocytes.

2. The enlarged liver and spleen, both exceedingly hard, with sharp and firm margins.

3. The marked difference in the number of colonies on blood culture from both internal jugular vein.

Jewish Hospital.

CHRONIC PURULENT OTITIS MEDIA AND ITS COMPLICATIONS.*

DR. ALBERT L. USSET, Chester, Pa.

Someone said that a person suffering from a chronic otorrhea is practically living over a volcano; that this is literally true, all otologists will agree, for nowhere in the human body do we find so many vital structures closely approaching each other as in the body of the temporal bone.

Mrs. A. B., white, age 30 years, came under observation on July 30 in the Nose and Throat Clinic. Her chief complaint was a chronic otorrhea and frequent attacks of otalgia. Her family history was unimportant.

P. P. H.: About eleven years ago she was treated in an industrial plant by a nurse for earache; since then she has had a discharge from the left ear with frequent attacks of otalgia. Examination of left ear showed a chronic purulent otitis media, membrana tympani absent, all landmarks absent, large polypoid mass over inner wall of middle ear. I advised her to have the poly removed, as it would facilitate drainage, and incidentally assist in obtaining a better view of the middle ear, and instructed her to return the following week.

I did not see her again until August 10, when I was asked to call at her home as she was very ill, and found her in bed suffering agon-

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izing pains. She complained of having pain in left ear and over mastoid region for the past three days, severe headache and insomnia.

Examination of ear showed same picture as on previous consultation, with the exception that the otorrhea was quite profuse and fetid, signifying bone necrosis. There was marked tenderness over the antrum and the tip of the mastoid. Temperature 101.° Pulse 96.

I removed the polypoid mass with a snare and ordered an instillation of boric acid and alcohol, also ammonal gr. V every four hours if required for the pain. During the day she was fairly comfortable, the pain disappeared, and she was even able to take some light food for dinner, but about 7 p. m. she suddenly became unconscious and remained comatose until admitted to hospital three hours later (10 p. m.). Shortly after admission to hospital she regained consciousness, replied to questions intelligently, though cerebration was somewhat retarded, pupils reacted to light and accommodation, no spontaneous nystagmus, did not remember anything that happened that evening prior to admission.

Examination showed distinct tenderness over the mastoid, especially over the sigmoid sinus, also over sternocleidomastoid muscle.

At 2:25 a. m., August 11, she had a chill lasting twenty minutes, temperature rose to 101° and then descended to normal.

The following morning the mental condition progressed to a mild delirium, temperature and pulse ascended gradually, and she complained of having chilly sensations, but had no distinct chill. B. P., 90 over 60.

Urinalysis showed a trace of albumen and the presence of sugar.

Blood examination: R. B. C., 2,700,000. W. B. C., 5,500. Hemoglobin, 78 per cent. Polymorphonuclear leucocytes, 69 per cent. Lymphocytes, 38 per cent. Mononuclears, 3 per cent. Blood culture showed no growth after five days incubation.

X-ray report: Sclerosis left mastoid, entire loss of cellular element, general opacity.

August 12, her condition became worse, temperature 103,° pulse and respiration increased proportionately, delirium became more marked and upon consultation with Dr. Orr, we decided to explore the mastoid. Operation performed by Dr. Orr consisted of a simple mastoidectomy; cortex was found sclerotic and filled with foul-smelling pus, suggestive of a colon bacillus infection or cholesteatoma. Lateral sinus was found about one-eighth of an inch more forward than normal; when uncovered it was found to be plastered over with a grayish layer of pus, showing the presence of a perisinus abscess.

The sinus was accidently opened and the hemorrhage controlled with tight packs.

After the operation the respirations became very slow; at 12 p. m., respiration 16, pulse 140, temperature 105.° At 5 a. m. she was delivered by the resident physician with a live baby without any laceration and very little bleeding. Respirations improved following the delivery, but soon after she became unconscious until death, which took place August 14.

Now the question naturally arises, did this patient have a sinus thrombosis or not? That it is a difficult question I am sure you will all agree with me. We have here certain symptoms which are suggestive of the presence of a sinus thrombosis.

1. The presence of a chill for twenty minutes.

2. High temperature and low leucocyte count. White says, "A high pulse rate and a comparatively low leucocyte count are the normal findings in sinus thrombosis."

3. The presence of chilly sensations. Macuen Smith says, "In the presence of an otorrhea, especially of the chronic type, the chilly sensations frequently spoken of with little thought of its significance is often the beginning of an intradural suppuration."

4. Extreme tenderness over the sinus, as well as over the sternocleidomastoid muscle.

5. Presence of a perisinus abscess.

On the other hand, we have here certain symptoms which do not point to the presence of a sinus thrombosis, the main one due to the fact that we obtained free bleeding upon opening the sinus. This, however, is not conclusive proof, as she may have had a mural clot.

328 East Broad Street.

HEMANGIOMA OF TONSILLAR FOSSA.*

Dr. G. B. Trible, Washington, D. C.

Case Report: Mr. M. G., age 30 years, white, single; business man with a negative family history. Previous history of pulmonary tuberculosis and a tubercular involvement of the seminal vesicles with consequent sterility. Tonsillectomy some years before—fossa, left, clear; right, with a large mass.

Present Illness: In the course of routine examination by an internist, a large mass was noted in the right tonsillar fossa, painless, the patient being unaware of his condition prior to this time. He was advised to let it alone pending further developments, and in this judgment his family physician, Dr. Norman Smiler, concurred. After about eleven months, it was noted that this mass was increasing in size and causing some difficulty in swallowing. Dr. Smiler advised him to consult a throat specialist, and later accompanied him for examination. Pharyngeal examination revealed a globular swelling in the right tonsillar fossa, presenting about the size of an English walnut, not pulsating, not movable, smooth, covered by mucous membrane. It had increased in size markedly in the past few weeks. There was no pain, and no fluctuation could be made out. No enlargement of corresponding lymphatics. Because of the active tuberculosis, a radiograph of the cervical vertebrae, with the possibility in mind of a tuberculous involvement of the glands communicating with a vertebral focus, was suggested. Radiograph was negative. Blood examination negative. Wassermann negative. Temperature normal. In view of the fact that the examination showed a suggestion of bogginess, he was advised to have a needle inserted in the swelling under sterile precautions, and an attempt made to remove some of the contents for examination, or if it were solid, to have a section removed for study. The patient was not satisfied with the lack of definite diagnosis; so he consulted several other specialists, one of whom felt it advisable to incise the mass, which was done and followed by profuse hemorrhage, necessitating the patient's throat being sutured, and only after considerable difficulty, the bleeding was stopped. He then went to Philadelphia and consulted another specialist, who went over the case thoroughly, but had no new suggestion except a possibility of a carotid tumor. He advised removal of a piece of the mass for bioscopy.

*Read before the Eye, Ear, Nose and Throat Section, District of Columbia Medical Society, Dec. 17, 1926.

The patient again returned to this office and wished this done. A few days later, at the Emergency Hospital, Dr. Smiler and the writer, under local anesthesia undertook to remove a section, but finding it possible by blunt dissection to detach the growth on all sides except posteriorly, and feeling that it was no more dangerous to attempt a complete removal than to remove a section sufficiently large for examination, continued dissection and removed the growth intact, except for a tear at the posterior point of detachment.

The apparent size, before operation, was about that of a large English walnut and extra-situ was about one-third or one-half of its former size. A mattress suture was placed across the line of denuded tissue and bleeding stopped without much difficulty. The growth was immediately placed in fixing solution and sent to laboratory for examination.

Laboratory Report: Tumor tissue—hemangioma, fibromuscular-myxomatous base—non-malignant. A slide was sent to Dr. Bloodgood for an opinion, and he verified Dr. Neuman's report.

For the first ten days following the operation, the recovery was about as uneventful as that following tonsillectomy. After that time no progress was made and an indurated mass developed at the back of the fossa, involving the posterior pillar, and looked decidedly malignant. He ran temperature, which was probably due in part at least to the tuberculosis, and it was decided to use either radium needles or X-ray exposures, with an idea of limiting any pathological changes. X-ray exposure was decided upon because of the ease of application and the non-interference with the nutrition, which radium needles would cause. Consultation was called and the line of procedure practiced approved of, and two exposures to X-ray were given. Crow advised by letter, radium, and if the case had not been tubercular, with poor nutrition, this would have been done and may, of course, be necessary later.

Discussion: Benign tumors of the pharynx and tonsil are comparatively rare. Of course, in this case, the tonsil tissue had been removed, probably thoroughly. One of the first cases of benign growths was that reported by Debaussy, followed later by P. F. Eve in the *Nashville Journal of Medicine and Surgery.* Hemangioma is one of the rarest benign tumors to occur in the pharynx. Its rarity is emphasized when it is recalled that Schmidt in a review of neoplasms does not report a single case.

Benign Tumors: Any of the ordinary benign neoplasms may occur in the pharynx or tonsil region. The most common is a fibroma or mixed type of fibromyxoma with fibrous elements predominating.

Myoma, myxoma, cysts, hemangioma and lymphangioma occur. Rarely a pure type tumor is found. Most commonly the structure is composed of several elements—for instance, fibromyxomyoma, which is frequent.

Causes: Where little is known, much is written, and to discuss the causes of tumor is to merely repeat and rehash that which has been handed down by others. There is a very strong predisposing factor which can always be found in the pharynx, more exposed as it is, to irritation than any other mucous-lined cavity in the body. The mechanical irritation incident to mastication and deglutition is more or less constant. Extreme temperature changes are suffered by no other mucous membrane comparable with those constantly suffered by the pharynx and oral cavity. Substances that are too hot or too cold to be tolerated by the skin are swallowed.

Infections: Infections are quite common, the mucosa being involved to a greater or lesser degree with every coryza, sinusitis, or nasopharyngitis, bathed in pus in the septic mouths, tanned by constant smoking and the wonder is that resistance has been developed to such a high degree that these tumors are so infrequent. Tumors of the pharynx are more common in the male—the age of election, that between twenty-five and forty—the symptoms usually due to mechanical influences. From the position and size they affect: *1.* Deglutition; *2.* respiration; *3.* the voice; *4.* the hearing; *5.* the circulation.

One of the first symptoms to appear is the sense of fullness and interference with swallowing. This interference with deglutition varies with the size of the tumor from an almost imperceptible beginning until the tumor progresses in size and is blocking more and more the oropharynx. Then there is an inability to swallow anything but fluids and even with these there is a tendency to regurgitation through the nose.

Second: As the tumor progresses in size there is interference with the respiration, due to the blocking of the upper air passages, and with this there is frequently a change in the voice, which may assume a nasal character.

Third: Another symptom is the loss of acuity in hearing, homolaterally. This is probably due to a congestion and blocking of the Eustachian tube and the consequent retraction of the tympanum from air absorption.

Fourth: If the tumor progresses in size, circulatory embarrassment, due to pressure on the jugular veins, is inevitable. Consequently there would be symptoms of a local venus stasis.

Fifth: Another very annoying symptom, which may be present, is a reflex cough due to disturbance of the epiglottis. With this there may be nausea, especially if the tumor is large enough to encroach much upon the pharynx.

These growths very seldom give pain, unless there is an associated inflammation of the pharynx and upper air passages, though a catarrhal condition is often observed, due to the constant irritation.

Angioma: Angioma as found to be one of the rarest of tonsillar and pharyngeal tumors. As before noted, Schmidt does not report one case in his treatise on benign tumors in the oropharynx. When it is extensive, it may even involve the larynx, and is described as frequently irregular in contour. It may or may not pulsate, and in color it varies from brownish to wine-red. Histologically, it is usually composed of venous elements. The new-formed vessels resemble normal veins and are interlaced with each other to form a cavernous mass.

In the case reported, the tonsillar fossa was completely filled, the mass extended superiorly behind the palate, and pushed forward the tissue over the last molar on the lower right jaw. The color did not differ markedly from the normal mucosa; it did not pulsate; it was regular and smooth in contour; and gave the appearance of a cyst. Symptoms from it were purely those occasioned by its size and position.

Treatment: The treatment is always surgical. Those most commonly advocated are ligation of the vessels, which causes a shrinkage and atrophy due to lack of nutrition.

Second: Electrolysis and cauterization, removing the tumor with the least possibility of hemorrhage.

Third: Excision, considered the most unsatisfactory, as it is frequently accompanied by extensive hemorrhage, difficult to control.

In this case dissection was not especially difficult and the hemorrhage was controlled and the success can probably be attributed to a lack of knowledge of the real nature of this growth, and its consequent treatment along purely surgical lines. The swelling and induration around the posterior pillar appears, in the light of later events, to have been due to disturbance of circulation and lymphatics, and is noted in the removal of hemangioma from other regions.

I am indebted to Dr. Smiler for his assistance and support, and to Dr. Constantinople for searching the literature relating to this subject.

1801 I Street, N. W.

VINCENT'S ANGINA.*

DR. LOUIS R. EFFLER, Toledo, Ohio.

There is no pretense at making this study exhaustive. Perhaps nothing new will be forthcoming. There will be no quotations from authorities nor reference to biliography. The author has purposely not gathered a selected number of cases to prove a point. He has had no regard for time, sequence or number of cases. He has purposely refrained from delving into his records. His one purpose has been to view the subject impartially and draw what lessons might suggest themselves. He has jotted down the first ten cases of Vincent's angina that have come to mind. Perhaps there was something noteworthy to recall them to memory. If so, no preconceived points of emphasis were planned. Let us briefly go over these cases and note what may be suggested by this study. It goes without saying that all these cases are proved by laboratory diagnosis.

Case 1: Mr. H., age 45 years. Nov. 3, 1925, illness began with symptoms of "flu." Nov. 13, called in consultation by family doctor. Patient was and had been very sick, with a temperature range from 101° to 103.° Patient was physically above par. A dirty-looking ulcer, covered with a typical "mealy membrane," involved the right tonsil. The tonsils were lymphoid and hypertrophied 4x. The gums were in bad condition and also involved. Further consultation was had.

Nov. 16, patient was removed to hospital because no improvement in local or general condition was noted. During the hospital stay, every effort was made to combat an associated septicemia. Blood culture revealed streptococcus. Despite injections of acriflavin, saline, salvarsan, antistreptococcus serum, transfusion, etc., in addition to local measures, patient lost ground daily. Temperature ranged from 98° to 107.°

Dec. 2, patient finally died.

Case 2: Mr. C., age 28 years. Oct. 1, 1926, patient was first seen after a two weeks' sore throat. Patient was physically above par. Both tonsils were involved with extensive destructive ulceration. The tonsils were the lymphoid type, hypertrophied 4x. Despite his youth, patient had had all his teeth extracted three years before. It was impossible to determine whether this was due to Vincent's angina involving the gums.

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Oct. 27, patient was discharged cured, following regular local treatment during the interval.

Case 3: Miss M., age 41 years. July 6, 1926, patient referred in consultation after being treated for several weeks by her own doctor. Complaint was of sore throat, left. Patient was distinctly below par physically. Examination revealed several white patches occupying crypts of both tonsils. No temperature. Preliminary diagnosis was "mycosis." Gums were suspiciously involved.

July 10 to August 1, tonsils treated locally with a view to removing them safely. This was thought necessary because they were the buried type, prevented from drainage by redundant anterior pillars. Patient was evidently absorbing because she was losing weight and feeling badly.

August 3, T. and A. performed.

Oct. 29, patient had still not reached full strength despite complete rest, tonics, etc., since operation.

Case 4: Miss D., age 35 years. Dec. 4, 1926, had sore throat, left.

Dec. 11, seen for first time in office, complaining of earache. Patient was physically above par. Examination disclosed ulceration involving small tonsillar tab at base of left fossa. The pain in ear was referred from this region. The tonsils had been removed by me on June 25, 1924.

Dec. 21, 1926, local treatment had sufficiently cleared the tab, so that it was removed under local on this date.

Dec. 30, patient again O. K., though local reaction seemed disproportionate to cause.

Case 5: Mr. C., age 22 years. Dec. 13, 1926, patient first seen with sore throat, left. Had been under treatment for tonsillitis continuously for one month previous. Patient was physically above par. Examination showed deep, ragged ulceration involving the left tonsil. The tonsils were lymphoid type, hypertrophied 5x. Glands in left of neck much enlarged.

Dec. 22, patient discharged to all intents and purposes cured. Local treatment alone was used.

Case 7: Miss D., age 22 years. Nov. 30, 1926, patient first seen. Had been treated for recurrent "gum boils" by several reputable dentists without much result. Patient states they finally admitted their helplessness. Patient was physically below par. On examination a "gum boil" was noted in the region of lower incisors. Otherwise the mouth seemed in good condition. Smears from this region and from the tonsils revealed Vincent's. The tonsils were ragged, lymphoid type, hypertrophied 3x. A tonsillectomy was considered necessary to break up the "vicious circle."

Dec. 9, following local treatment in the interim, patient was deemed ready for T. and A. A last minute smear of gums and tonsils, however, revealed organisms so numerous that operation was postponed. Treatment was continued locally with Fowler's solution and generally with salvarsan injections.

Dec. 18, smears showed only occasional organisms every second or third field. T. and A. performed. Recovery uneventful.

Case 6: Mr. B., age 21 years. Dec. 6, 1926, sore throat, left, for past two days (?). Working out of Cleveland as a contractor. Has been living from hand-to-mouth for past few weeks. This may account for picking up infection. General physical condition above par. Examination showed deep ulceration of left tonsil. The tonsils were lymphoid type, hypertrophied 4x.

Dec. 11, following regular local treatment in the interval, patient discharged, to all intents and purposes cured.

Case 8: Miss M., age 19 years. Dec. 10, patient first seen with bad earache, left. A "wisdom tooth" on same side had been extracted by a qualified dentist one month before. The dentist had remarked about "trench mouth" existing at the time. Patient has been in much discomfort ever since, due to sore throat, left, and earache, left. General condition far above par, although patient complains that inability to eat is weakening her daily. Examination showed tonsils had been removed several years ago. A ragged, fairly deep ulceration involved the base of the tongue, left, and extended into the base of the left tonsillar fossa (lymphoid tissue?).

Dec. 15, local treatment in the interval made very little impression.

Dec. 16, began treatment with Fowler's solution locally, and salvarsan generally.

Dec. 20, symptoms relieved and throat condition entirely cleared.

Case 9: Mrs. S., age 36 years. Dec. 8, 1926, patient first seen with sore throat of some weeks' duration. Patient has been under treatment for bad teeth and gums since first childbirth, ten years ago. Treated by two reputable dentists. The tonsils were removed several years before this. Examination showed multiple (five) ulcerations involving the soft palate and stubs of tonsils. The gums were notably in bad condition. Patient was below par physically.

Dec. 9, began local treatment with Fowler's solution and general treatment with salvarsan.

Dec. 20, mouth condition cleared. Patient ready for extraction of lower left second and third molars. These seemed bad offenders.

Case 10: Mrs. D., age 72 years. Oct. 25, 1926, first saw patient in consultation for sore throat. Two teeth had been extracted three

days before. Patient had been slowly recuperating from an auto accident some nine months before, in which she received broken ribs and a severe shaking up. On examination it was evident, considering age and accident, that patient's general condition was well above par. Disposition surprisingly cheerful in the face of bodily discomfort. Temperature 103.° Examination noted dirty, grayish membrane covering two above-mentioned tooth sockets and similar membrane covering both tonsils.

Oct. 28, after three days of local treatment at home, patient was sent to hospital. It was obvious that the general physical condition was becoming progressively worse, due to a strep. septicemia.

Oct. 30, despite treatment of all kinds, patient died.

CONCLUSIONS.

1. *Age:* Cases before 20 equals one; in 20's equals four; in 30's equals two; in 40's equals two; in 70's equals one. This would indicate that the disease is commoner in youth between the ages of 20 and 40. It is also more severe later in life. Note cases aged 70 and 45 died. Case at 41 had prolonged recovery.

2. *Physical Condition:* Cases in this group above par numbered six; below par, four. This at least refutes the notion that Vincent's agina attacks only the weak and debilitated. It also attacks the strong. By a peculiar coincidence, two deaths occurred in this latter class. These happened also to be the oldest (70 and 45). The worst that happened at 41, who was below par, was to prolong recovery.

3. *Acuteness or Chronicity:* Acute cases numbered eight; chronic cases, two. This has reference only to the time symptoms were first noticed. It cannot say whether the condition was present for any length of time previously.

4. *Tertiary Infections:* It is commonly supposed that Vincent's angina is a secondary infection. In other words, it attacks only when local tissues are traumatized or reduced in resistance by a primary infection. We cannot prove this from these cases. We do note, however, that in four cases a marked flare up followed immediately on trauma in its presence. We doubt whether Vincent's angina alone is ever sufficient to kill. The assumption must therefore be made that a tertiary infection is responsible for the severe systemic reactions and even occasional deaths. In two cases of this study, death was due to streptococcus septicemia. Incidentally, it is providential that so few deaths occur. With the natural defenses down, due to a break in the continuity of the mucous membrane, it becomes a question only of how virulent is the tertiary invader or how depleted the resistance of the host.

5. Tonsils or Gums: There was always a supposed predilection in Vincent's angina for lymphoid tissues. Here we note the tonsils alone were involved in four cases. In one of these, there were no teeth. In another, there was only a small tonsil stub no larger than a pea. The gums were involved alone in only one case. Peculiarly, in this case there were no tonsils. Both the tonsils and the gums were involved in five cases. In one of these, the tonsils had been removed, leaving several small tabs, which were involved, in addition to multiple ulcerations of the soft palate.

This overlapping of tonsils and gums obviously makes Vincent's angina no longer a problem for only the pharyngologist or only the dentist. At least in 50 per cent the two are involved together. When treatment is given from only the one angle, it may prove inadequate. Reinfections may take place. The "vicious circle" of gums to tonsils and tonsils to gums is too inviting. It is time for collaboration between the dentist and the pharyngologist and the pathologist.

6. Treatment: Vincent's angina of the tonsils alone responds to topical applications of any respectable germicide. At least, frank ulcers seem to yield readily to copper sulphate, tincture of iodin, silver nitrate in bead or in solution, chromic acid, trichloracetic, salvarsan, etc. Each has its ardent advocates. The organisms are not particularly resistant. We presume this is also true of frank lesions of the gums. But where any surgical measures are contemplated, v. g. tonsillectomy, teeth extraction, etc., I should not be satisfied with these measures alone. I cannot conceive that any of the above germicides will penetrate to every tonsillar crypt, especially in a buried or semiburied type, nor to every gingival fold, especially in a partly erupted third molar. I should prefer in combination with local treatment the injection into the blood stream or muscle of a known spirillum killer like salvarsan. This worked out very satisfactorily in three cases of this series where local measures alone were not sufficient. It is obvious that salvarsan will not affect a streptococcus septicemia.

SUMMARY.

In summary, Vincent's angina is not to be scorned as the harmless disease we once thought. It is capable of contributing to much suffering, grave danger, and even death. These are the points that should be stressed: Look for Vincent's angina before attempting any mouth surgery. Clean it up by local and general medication as above indicated. An ante-operative laboratory smear can be made as routine as a coagulation test.

222 Michigan street.

TRAUMATIC MASTOIDITIS WITH POSTOPERATIVE SIGNS SUGGESTIVE OF AN INTRACRANIAL COMPLICATION.*

DR. WALTER L. HORN, New York City.

H. S., boy, age 6 years, was struck by an automobile and was admitted eight hours later to the surgical service of the Mt. Sinai Hospital. There it was noted that the right ear was bleeding profusely and that there was no apparent escape of cerebrospinal fluid and no bleeding from the nose or mouth. At no time did the child lose consciousness, but was somewhat drowsy for about two hours directly after the accident. He vomited seven or eight times and complained of headache and pain in the right ear.

On admission the child was found to be wide awake, but restless. He showed no evidence of external violence, no swellings, no bruises. The pupils were equal, reacted well to light and aside from a converging strabismus, which was congenital in character, there were no objective neurological findings. The temperature, pulse and respiration were normal.

An examination of the right ear revealed a linear tear in the membrana tympani in its upper and posterior quadrant through which bloody fluid was oozing. Hearing, however, in that ear was normal.

An X-ray examination of the skull was not entirely satisfactory due to restlessness of the patient, but still the report read: "No visible fracture noted."

The first eight days of the patient's stay in the hospital were uneventful; the temperature, pulse and respiration remained normal. He was comfortable, did not complain of anything, and was only occasionally drowsy. But on the eighth day there occurred an abrupt change, the temperature suddenly rose to 102° and continued to rise to 103.° He began to complain of severe pain in the right ear and a foul-smelling purulent discharge from the right ear was noted and there appeared a definite sagging of the posterior canal wall with edema and tenderness over the right mastoid. Hearing became somewhat impaired. The diagnosis of acute mastoiditis was made.

A simple mastoid operation was performed. At the operation a

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linear fracture was found. Its upper limits could not be ascertained, but beginning about 1 c.m. above the floor of the middle fossa in the squamous portion of the temporal bone the fracture extended downward vertically through the floor of the middle fossa across the mastoid bone and through the posterior canal wall just above the semicircular canal. There was some free pus and a breaking down of the mastoid cells. A free fragment of bone, apparently a portion of the floor of the middle fossa, was disclosed and removed. The overlying dura appeared normal. A subsequent bacteriological examination of pus from the mastoid showed streptococcus hemolyticus.

The postoperative course was uneventful for a period of twelve days. On the twelfth day a sudden change took place, the boy complained of headache, became irritable and vomited on two occasions. The temperature rose to 100,° the pulse to 100, and there developed mild meningeal signs, such as slight rigidity of the neck and bilateral Kernig. In addition there were found a definite left Babinski, hyperactive knee jerks and markedly diminished abdominal reflexes on the left side. Within a few hours there was elicited bilateral ankle clonus, which was more marked on the left side. These signs indicated a right focal lesion and in view of previously established suppurative focus in the right ear the possibility of cerebral abscess in the temporal lobe was considered.

A lumbar puncture yielded clear cerebrospinal fluid under markedly increased pressure. There were 88 cells, all lymphocytes, but no organisms on smear or culture. The neurologists favored the diagnosis of a circumscribed form of meningitis and considered surgical interference inadvisable, but the progressive advance in the condition of the patient continued, the ankle clonus became inexhaustible on the left side and the optic discs showed definite swelling. The question of a temporosphenoidal abscess was raised again, but the neurologists did not change their opinion and advised against operation.

The child was not operated upon. The conservative stand in the treatment was fullly justified by the subsequent course of events. Five days after the onset of the acute meningeal symptoms there occurred a turn for the better. The patient showed gradual but constant improvement, the meningeal signs subsided, the organic neurological signs disappeared, so that seven weeks after the injury the child was discharged from the hospital very much improved. Recently he was re-examined and found free from cerebral signs and symptoms and is regarded as having made a complete recovery. It is rather unlikely that there should be a latent brain abscess without sign or symptom.

Should I be permitted to speculate somewhat on the character of the lesion which precipitated the meningeal and focal cerebral signs, I would have suggested the following possibility: You will recall that at the operation a defect was found in the floor of the middle fossa. A piece of bone was removed at this point. Is it not possible that the traumatized dura with its overlying soft meninges became involved in a circumscribed aseptic meningeal process, which gave rise to the transient neurological signs and symptoms as noted in this case?

1040 Park Avenue.

A NEW OPERATION FOR EPISTAXIS.

Dr. George E. Davis, New York City.

Epistaxis is due to local and constitutional causes, for the most part local.

The seat of bleeding in 90 per cent of the cases is situated on the anterior part of the septum, about 2 c.m. above the nasal floor, and known as Kisselbach's area. The origin of the hemorrhage is from a plexus of vessels formed by the anastomis of branches, posteriorly from the sphenopalatine—or "artery of epistaxis"—above the ethmoidal, and below and anteriorly from the palatine.

Occasionally the point of bleeding is higher up and further back on the septum and the source of the hemorrhage is from the ethmoidal veins or telangiectases of other vessels. Usually in these cases the bleeding is profuse and intractable.

Orinarily cases of nose bleed are easily controlled. Rest, pressure, cold compresses externally, compresses of adrenalin at point of bleeding, 10 per cent solution silver nitrate on cotton-wound applicator or the galvanocautery generally afford prompt relief. But, it is different sailing when we encounter a desperate case—patient bled pallid, restless, respiration shallow and pulse thready.

Several operations have been proposed and practiced to check the bleeding and heal the local condition causing it. Leshure has reported a number of cases in which he was successful in checking the hemorrhage by elevating the mucous membrane, as in the submucous septum operation. Hunter Mackenzie, where the bleeding is profuse and from an extensive area, ablates part of the mucous membrane.

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However, as all remedies and procedures failed me in a case of extreme epistaxis, in the person of a husky blacksmith, who suffered from recurrent attacks at intervals varying from several days, weeks or months, in which the profuse bleeding seemed to continue until he was all but exsanguinated, or the heart became too weak to force the flow, the simple operation outlined below brought prompt and permanent relief and cure.

It occurred to me if I could locate and isolate the bleeding area I would be master of the situation.

After cleansing the nose of clots and shrinking the tissues with cotton compresses soaked in adrenalin and cocain, I observed the blood welling from a point high up and rather far back on the septum coming from a dilitated, broken ethmoidal vein. Under bright illumination, with a small right-angled septum knife, I made a vertical incision through the mucous membrane, one-half inch posterior to the bleeding area, another vertical incision about the same distance anterior to the bleeding area and with a straight blade made similar incisions above and below the bleeding area—being careful to make the upper and lower incisions nick or join the vertical cuts. These incisions must be far enough from the pathological or bleeding point or area to be made in normal tissues and extend not only through the mucous membrane but the perichondrium or periosteum as well. Bleeding from cuts in healthy mucous membrane is readily controlled and, of course, with the supply cut across in every direction, the bleeding area is isolated and the flow ceases immediately. The operative procedure is completed by cleansing the nose of clots, applying a 2 to 4 per cent silver nitrate solution on cotton-wound applicator to the original bleeding area and the fresh cuts as well. Packing the nose is not required—a pledget of cotton in the anterior nares, to be changed when soiled, is sufficient. Further local after-treatment consists in gentle cleansing and mild silver applications till healing is complete.

Of course any constitutional contributory causes should be corrected.

The epistaxis is permanently relieved since the future blood supply to the original area must come from small vessels entering through the *scar* tissue of the four isolating incisions in the mucous membrane.

The above operative procedure has never failed in other cases equally as severe as the one reported above.

42 West 77th Street.

IN MEMORIAM.

DR. WILLIAM SWEENEY STUCKY.

As we go to press we are in receipt of the sad tidings of the sudden death from cerebral hemorrhage of Dr. William Sweeney Stucky, of Lexington, Ky.

Dr. Stucky, the son of Dr. and Mrs. J. A. Stucky, was born in Lexington, February 8, 1884. He was a graduate of Bethany College of West Virginia, B.A., 1904; a graduate of the University of Michigan, receiving his degree in medicine there in 1908. He then spent two years as interne in the Post-Graduate Hospital, in New York City, followed by an internship at the Manhattan Eye and Ear Hospital, qualifying for the special practice of Ophthalmology and Otolaryngology.

On his return to Lexington, in 1911, he associated himself with his father in special practice and was active in local, state and national organizations and at the time of his death was president of the Fayette County Medical Society of Kentucky. He was a member of the Academy of Ophthalmology and Otolaryngology.

Dr. Stucky is survived by his widow, formerly Miss Annie Clay McDowell, of Lexington, a son and a daughter, and by his parents, Dr. and Mrs. J. A. Stucky, all of Lexington; two sisters, Mrs. Harold Williamson, of Lexington, and Mrs. A. B. Chapman, of Myrtle Point, Ore.; a brother, Harry C. Stucky, of Fort Myers, Fla.

Will Stucky was a universal favorite and his individual charm and fine spirit was apparent to everyone with whom he came in contact.

To his immediate family we extend our heartfelt condolence and to our dear friend, his father, we especially offer our deepest sympathy for this irreparable loss. Only recently those of us who were more intimately informed of his active professional co-operation with his esteemed father realized what a help and support this younger scion of the Stucky family had become.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

(Continued from page 386)

Another question is that of lateralization. If you take two tuning forks which are slightly different in pitch and hold one to each ear, you will get a very strange sensation; it will give the impression of a sound wandering around the room; but if you make the intensity in one ear 60 sens more than in the other, you will hear ordinary beats. The strange thing is that although we know the beat must be created in the ear which is receiving the weaker sound, the lateralization is always in the ear which is receiving the louder sound. So, there are many things about lateralization that are very deceptive. The phenomena may be referred to one ear, the actual physical fact may be in the other.

The question of testing unilateral deafness was discussed. It was a rather important fact for telephone engineers to learn that when you listen to the telephone in a noisy room you are not annoyed because the noise comes into the free ear, but because of the noise that leaks through into the ear against which you are holding the telephone receiver. It is possible to introduce noise into the free ear without interfering with hearing in the other ear as long as its intensity is not raised above 60 SU. That is very important to know. With an instrument such as the 3-A Audiometer, you can always introduce the noise at the proper level so that it will mask all weak sounds coming into that ear without changing the acuity in the other ear. This fact makes it possible to use noise for temporarily blocking the hearing in a much more intelligent way.

One point occurred to me while Dr. Fowler was speaking. When we first developed this bone conduction receiver I had a friend who was a physicist who was very hard of hearing, and consequently could report accurately what he heard.

The air conduction audiogram showed a loss of 50 SU for frequencies up to 4,000; then it suddenly dropped to low values. The bone conduction audiogram was slightly above normal for frequencies below 4,000 and suddenly dropped to low values for frequencies above this value. It seems safe to conclude from such curves that there was some impairment of the conduction apparatus which produces the lowering of acuity by air conduction with no impairment of the nerves except with about the first 8 m.m. of the basilar membrane where there is a nerve lesion. This shows the importance of making a complete audiogram of both air and bone conduction before a safe diagnosis can be made.

DR. GUTTMAN: After listening to the excellent presentation of the subject of hearing tests by the readers of the papers, it is rather difficult to add anything of interest or importance. I will therefore occupy the time at my disposal with a few elementary statements pertaining to the subject under discussion.

In the discussion of hearing tests, two factors have to be considered: 1. the sensation which is called hearing; this must be considered from the physiological and psychological viewpoints; 2. the cause of this sensation, which is sound; this must be studied from the point of view of the physicist.

Sound, as we know it, is produced by the vibration of the molecules of an elastic body, usually the air. The physicist is able to measure the amplitude of these vibrations, and he calls this the intensity of the sound; he is able to count the number of the vibrations per second—he calls this the frequency or pitch of the sound. The timbre, or quality of the sound can be left out of consideration for the present. While sound from the physicist's standpoint is fairly well understood, its effect, which is hearing, is from the physiological and pathological standpoint far less understood.

In order to understand hearing, which is the specific function of the normal and diseased ear, we must be able to measure this function. We must find out what is the least amount of intensity which is necessary to evoke the minimum of sensation—this is called the threshold of perception. The function of the diseased ear we measure by determining the increased amount of intensity which is necessary to produce the threshold of perception.

The old hearing tests were crude and inexact, because the unit of measurement was variable; the intensity of the voice, watch, tuning fork, Galton whistle, changed from examiner to examiner, and even with the same examiner from time to time. The audiometer, with which we can produce a sound which is uniform and stable at all times as regards intensity and pitch is therefore a great step forward. Whereas in the old hearing tests we applied as unit of measurement a yardstick which became elongated or shrunk from examination to examination; in the audiometer we possess a unit of measurement which is always stationery with regard to the intensity and frequency of the sound.

But with all that we must recognize its limitations: 1. With the audiometer we are measuring the hearing power of the patient for a tone of a pure, simple sound wave, as this can be done easiest and with great exactness; it is, however, not always easy to establish the relation between the perception of the tone and the perception of a very complex sound or noise, and the patient is primarily interested in determining his hearing capacity for speech, which is a noise. In nature, the ear has hardly ever the opportunity to listen to a tone of a pure sound wave, as almost all sounds reaching the ear are of a very complex nature. This may explain the discrepancies found between hearing tests made with the audiometer and with other means. 2. Several years ago a friend to whom I showed my audiometer (which at that time I called electric acumeter —as philologically audiometer is not correct—*audio* is Latin and *meter* is Greek) asked me: "Do you gain any additional information regarding the pathological condition, the seat of the disease, or the treatment of the disease?" I must repeat today the same answer I gave him then: "No."

From a theoretical standpoint the audiometer has great possibilities; from a practical point of view, however, its chief advantage lies in the fact that we can with one glance visualize from the audiogram the hearing power of the patient, just as we can observe with one glance the temperature of the patient from the temperature chart.

With all that, however, the invention and introduction of the audiometer is a great step forward in otology. The advances in many branches of science were preceded by the invention of new instruments and new methods of examination. The telescope preceded the advance made in astronomy; the microscope, he advance made in bacteriology; the ophthalmoscope, the advances made in ophthalmology. We are therefore justified in expecting that the audiometer will be the forerunner of the advances which will be made in this branch of otology.

DR. SONNENSCHEIN: I wish to thank you for the kind reception you have given me and for the discussion. With regard to the loss of high tones, I would like to emphasize what Dr. Fowler said. We are all surprised at times to learn in acute otitis media that the loss of high tones is very marked, and it is hard to explain unless we assume that the inner ear is involved by wav of the round window. After the otitis subsides, the high tones usually return.

I am very greatly in favor, as most of the men here are, of having instruments of precision and the audiometer is of this type. I further believe in accurate measurements and this is possible with such an instrument. There are, however, a few objections. In the first place, the expense is great; secondly, unless the new attachment for bone conduction proves successful, it is not possible with the type of audiometer now largely used to properly test bone conduction; and I feel bone conduction is the key to diagnosis and prognosis in many instances. Furthermore, with the 2A Audiometer, we cannot test below 64 double vibrations nor above 8,192 double vibrations. On the other hand, there is no question that the audiogram is a most valuable permanent record for making comparison of former examinations or with the records of other otologists. However, it is not always possible to make a com-

plete diagnosis from the curve of the audiogram, and the clinical data, together with tuning fork findings, is usually desirable. With tuning forks of good quality when provided with the constant of damping or decrement, it is possible to get an audiogram similar to that of the audiometer, the only difference being that if a number of pitches are tested, the time consumed is greater than with the audiometer.

The reason that Dr. Minton and myself developed the method of testing both air and bone conduction with the stem of the fork was to overcome the theoretical objections offered by many men because of the fact that when testing air conduction it was done by the transverse vibrations of the prongs, while the bone conduction was tested by the longitudinal vibrations of the stem of the fork. So far as the transmission of sound through the tubing was concerned, we knew that while sound is best transmitted through solids, less through fluids and least well in gases such as air, that refers to sounds that are generated only in the medium in which they are heard. When the sound passes from one medium, such as a gas, into a fluid one, there is not a good transimssion of the sound. However, when the stem of the fork is placed in the tubing, the sound is conducted to the ear through the air column in the lumen of the tube and very little, if any, sound waves pass through the wall of the tubing.

So far as lateralization of sound is concerned, especially with reference to the newly-described or rediscovered cranial resonance tests, one may say that the question of lateralization is always a difficult one and that many individuals cannot appreciate a lateralization; and also the lateralization sometimes goes to the side contrary to the one that would be expected from the nature of the lesion.

DR. FOWLER: It might be well to caution once again as to the fact that there is no such thing as pure air conduction or pure bone conduction. When we speak of air or bone conduction, we are speaking relatively. All sounds come by both air and bone, and you cannot avoid this. Therefore, bear in mind that these tests mean only relative predominance of air conduction or bone conduction, and not hearing purely by either.

As to the different frequencies, we are very much in the dark as to what interpretation to assign to their connotations, but by repeated tests, checked up by tuning forks, we can say at least these are accurate, and that already a little light is beginning to come through regarding their meaning. With the help of the physicist to keep up straight, I believe that by means of the audiograms and air conduction and bone conduction, we will be able to say that in such and such an ear such and such a thing is happening; and if we can say that, we will be making a better diagnosis. In the past we have said many things which are not true. It does not follow that because we say Rinne is negative or positive that this means true nerve or obstructive deafness; but a definite statement that bone conduction is up or down so much is certainly a more accurate statement of a pertinent fact.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, with the Academy Meeting Deferred to Feb. 3, 1927.

Demonstration of Specimens of the Sphenopalatine and Gasserian Ganglia. Dr. Julius I. Klepper.

The Symptomatology of the Nasal Ganglion. Dr. Simon L. Ruskin.

Some Observations on the Cranial Sympathetic. Dr. Frank Henry Pike.

An Approach to the Problem of Pain Fields, Especially Those Related to Nasopharyngeal Disease. Dr. Frank Henry Pike.

The attempt to account for the pain in regions rather remote from the site of the primary disease process on the basis of pure referred pain has proved fruitless in some conditions. Too many unknown and mysterious influences enter into these attempts to allow of any serious consideration of referred pain

as a general explanation, and it is especially faulty in affording a basis of explanation for pain in the neck or back in cases of nasopharyngeal disease. The study of the fields of pain associated with metastases from uterine or prostatic neoplasms shows that the areas of pain may be explained by the involvement of spinal nerve roots by metastatic growths which occur in or close to the lymphatic nodes receiving the lymphatic drainage of the pelvic viscera. The known relations of the lymphatic glands of the neck, into which the lymphatic vessels of the nose and pharynx drain, to the nerve trunks in whose peripheral distribution the pain is felt, suggest strongly that the pain is due to irritative processes arising in the cervical lymphatic glands. The occurrence of enlarged and sensitive glands in the neck at the time the pain is felt in the neck, back of the head or shoulders, is corroborative evidence of the correctness of this view.

DISCUSSION.

DR. LOUIS HUBERT: If you want to get a correct description of the sphenopalatine ganglion consult the physiologist and not the anatomist. There is really only one anatomist, Professor J. Parsons Schaeffer, of the Jefferson Medical College of Philadelphia, who has given particular attention to the anatomy of the nose, and who has correctly described this ganglion. I do not understand why rhinologists fail to quote him when writing on this subject.

The best paper written on the sphenopalatine ganglion was by Mueller and Dahl in 1910, who for the first time demonstrated histologically this ganglion in man. They have clearly shown that it is a pure sympathetic ganglion.

What are some of the characteristics of the sympathetic ganglia and the sympathetic nervous system?

1. The sympathetic ganglionic cells are multipolar, in contradistinction to the unipolar cells of the sensory ganglia.

2. Each sympathetic ganglion receives medullated nerve fibers which have their origin in the lateral horn cells of the spinal cord or in the corresponding regions of the medulla and midbrain. These nerve fibers are called preganglionic and constitute the motor or excitor root of the ganglion. This motor root takes its course through a motor nerve.

3. From the sympathetic ganglion other nerve fibers, called postganglionic, are given off, which are usually nonmedullated and which proceed from the ganglion to the structures they supply. When these postganglionic fibers have a long way to travel to reach their destination, they enter sensory nerves or accompany blood vessels.

4. The function of the sympathetic nervous system is to regulate the caliber of the blood vessels, to control glandular secretion and to act on the involuntary muscles in general.

5. The sympathetic nervous system is not in itself capable of reflex reactions. Impulses must arrive from the cerebrospinal nervous system, through afferent somatic nerves.

If the above physiologic principles were kept in mind, the medical literature would be free of the many errors and nonsense written about the sphenopalatine ganglion. The fault is not so much with the writers as with the textbooks of anatomy, where we would expect to find a correct description of the ganglion. But these textbooks have been written before the histology of the ganglion was worked out. They describe the ganglion from the gross anatomical point of view, which is very confusing. They tell us that the ganglion has three roots: a sensory, a sympathetic and a motor root. Let us analyze these roots. The sensory root is said to consist of the sphenopalatine nerve or nerves, connecting the maxillary nerve with the upper border of the ganglion. These so-called sensory root fibers, however, have no connection with the cells of the sphenopalatine ganglion. They pass around or through the ganglion, sending a few filament to its capsule. The so-called sensory branches of the ganglion are in reality continuations of the second division of the fifth cranial nerve. We may, therefore, eliminate the so-called sensory root.

Now let us consider the so-called sympathetic root. This is said to be formed by the large deep petrosal nerve of the carotid plexus. This nerve is made up of postganglionic fibers that are given off by the superior cervical ganglion. According to a physiologic law worked out by Langley and con-

firmed by others, these postganglionic fibers cannot be interrupted again in another ganglion. Therefore they have no connection with the cells of the sphenopalatine ganglion, but pass around it and enter the sensory branches of the second division of the fifth cranial nerve, to be distributed to the glands and blood vessels. Inasmuch as some fibers of the carotid plexus are medullated, it was thought that they represent preganglionic fibers coming from the lateral horn cells of the first to the fourth thoracic segments of the spinal cord, that they pass through the cervical ganglia uninterrupted, end in aborizations around the cells of the sphenopalatine ganglion, and so constitute the sympathetic root. It has, however, been shown by Ranson in the *Journal of Comparative Neurology*, in 1918, that some of the postganglionic fibers given off by the superior cervical ganglion may and do acquire a medullary sheath. So we have eliminated the so-called sympathetic root.

Let us finally consider the motor root. It is made up of preganglionic fibers which have their origin in the medulla near the nucleus of the nerve of Wrisberg, enter the racial nerve, leave the latter at its knee, where the geniculate ganglion is situated. Von Lenhossek has shown that these preganglionic fibers have no connection whatsoever with the geniculate ganglion, that they become finally a component fiber of the Vidian nerve and reach their termination by aborizing around the cells of the sphenopalatine ganglion. Gaskell calls this motor root the large superficial petrosal nerve. Other anatomists, like Schaeffer, designate as the large superficial petrosal nerve those sensory fibers that have their cells bodies in the geniculate ganglion. The latter nerve fibers, however, have no connection with the cells of the sphenopalatine ganglion, as wrongly stated by Dr. Ruskin, but pass through the ganglion without interruption and are distributed through the so-called ganglionic branches. Again, within this motor root are real motor fibers that go through the ganglion also without interruption, and supply the azygos uvulae and the levator palati muscles.

We can now easily understand the pure sympathetic nature of the sphenopalatine ganglion. It is best to forget the gross anatomical description of its roots, which only causes confusion. Like any other sympathetic ganglion, it receives preganglionic fibers that take their course through a motor nerve (in this instance the facial nerve), and sends out postganglionic fibers that are distributed to the glands and blood vessels by means of the so-called ganglionic branches, which, as said above, are in reality a continuation of the second division of the fifth nerve. This ganglion selects the best position available for the distribution of its postganglionic fibers, i.e., where the second division divides in its various branches. It is merely a question of human economy.

If it is true that the sphenopalatine ganglion is a pure sympathetic ganglion, then the statements made in textbooks that it gives off sensory branches to the nose, hard and soft palate, and tonsils, and motor fibers to the azygos uvulae and levator palati muscles are incorrect. A sympathetic ganglion does not send out fibers belonging to the voluntary nervous system.

It is also incorrect to say that cocain anesthetizes the sphenopalatine ganglion, if it is a true sympathetic ganglion. We know that cocain stimulates the sympathetic nervous system and that it has a selective action on sensory nerves, which it paralyzes. Therefore, when cocain is applied to the sphenopalatine ganglion the branches of the second division of the fifth nerve as they surround the ganglion are anesthetized. It is impossible to apply cocain to the sphenopalatine ganglion without at the same time applying it to the sensory nerves that surround it. Therefore, the relief of the symptoms supposedly attributed to the action of this drug on the ganglion is, in all probability, due to its effect on the sensory nerves. Again, the injection of the ganglion with novocain and alcohol through the posterior palatine canal as described by Ruskin is not an injection of the ganglion, but of the second division of the fifth cranial nerve. As a matter of fact, this is one of the methods of injecting the second division of the fifth nerve taught at the Vienna Medical School. Furthermore, let us suppose that cocain can be applied directly to the sphenopalatine ganglion and relieves certain symptoms. This does not necessarily mean that injection of the ganglion would do the patient any good, as the relief of the symptoms might be explained by a stimulation of the ganglion by the cocain.

What are the symptoms in rhinology which can be attributed to a disturbance of the sphenopalatine ganglion?

Rhinorrhea, sneezing and lacrimation may be caused by an irritation of the sphenopalatine ganglion. The mechanism of these symptoms I have fully described in a paper read before the Nose and Throat Section of the New York Academy of Medicine in 1921. There is another symptom; namely, pain, which Sluder has attributed to an involvement of the sphenopalatine ganglion per se. I believe, however, with Professor J. Parsons Schaeffer, that most of the pains described by Sluder and supposedly associated with a disease of the nasal ganglion, are in reality an expression of irritation of the peripheral fibers of the trigeminal nerve, which pass via the ganglion in their course centrally. Overflow of the afferent sensations would cause them to be referred along certain other peripheral trigeminal fibers and probably along other nerves with which the latter establish peripheral communications. However, pain referred to regions far removed from the distribution of the trigeminal nerve also occurs and its explanation must be sought in a disturbance of afferent sympathetic neurons. Whether or not such fibers exist in the nasal cavity has not been established as yet. From analogy and the clinical evidence at hand it is assumed that such fibers are present and that they have their cell bodies in the geniculate ganglion, the first and second thoracic, and possibly also in the cervical posterior root ganglia. By assuming the existence of these fibers we can explain the remote distribution of the pain in nasal disease. It is not necessary to assume that the sphenopalatine ganglion is partly sensory. Sympathetic pain is due to irritation of the sympathetic afferent fibers which carry the pain impulse to the somatic sensory (posterior root) ganglia. The transferred impulse may then be referred to the peripheral endings of the somatic sensory neurons coming from the somatic sensory ganglia and interpreted by the brain as pain.

In conclusion, I wish to reiterate that there is very little, if any, anatomical or physiological basis for the belief that the sphenopalatine ganglion has anything to do with the causation of pain. This, however, does not exclude the existence of sympathetic pain in nasal disease. As said above, sympathetic pain is due to irritation of afferent sympathetic fibers, and it is not necessary for the irritation to be in the region of the sphenopalatine ganglion. I wish also to warn against indiscriminate injection of alcohol in the region of the ganglion. This should not be done unless the patient has been thoroughly examined by a competent neurologist and a psychoneurosis excluded. I know patients whose symptoms have been relieved after injection of the ganglion. But upon the return of the symptoms another injections failed to give relief.

This critical analysis does not in any way belittle the original and wonderful work done by Dr. Sluder. As a matter of fact, after reading his book about nine years ago, I was stimulated to take up the study of the sympathetic nervous system. In Dr. Sluder's writings you can easily distinguish his facts from his speculations and theories, but the trouble is that most men who have written on this subject have referred to Dr. Sluder's speculations and theories as facts.

DR. W. G. PENFIELD: Dr. Pike pointed out different types of neuralgia in the head: First, trigeminal, which is, of course, relieved by section of the posterior root of the fifth nerve. He also pointed out that we do not know the cause of trigeminal neuralgia and many examinations of the trigeminal ganglion has been made and reveal nothing. In spite of that ignorance we can and must relieve these patients of their pain, especially as it can be done so easily and safely by section of the sensory root. Too many patients are allowed to suffer needlessly for years.

A second type of neuralgia is marked by pain in the ear, and this has been pointed out by Adson to be a glossopharyngeal neuralgia. It has been relieved by him in a number of cases by section of the glossopharyngeal root within the skull.

Dr. Sluder has pointed out this third type of neuralgia. In cases of supposed trigeminal neuralgia which we see and which are not quite typical, we have learned to consider this type of pain.

I think there is great danger in following out too far any one particular line of therapy. We have tonight been shown very clearly the anatomy of the sphenopalatine ganglion; but, as Dr. Hubert has pointed out, we must also bear in mind the physiology of the autonomic nervous system. There is only one type of reflex confined to the autonomic nervous system. This is the so-called Sokownin reflex. It is an axone reflex which occurs with the collaterals of one ganglion cell not pressing on the others. Other types of autonomic reflex involve the central nervous system as well.

Golz' dog from which the brain had been removed down to the optic thalmus was able to walk about and had all of his autonomic nerves intact, as well as his cranial nerves. The odor of food did not make him salivate nor did the smell of another dog produce any of the results Dr. Ruskin has mentioned as due to reflexes within the autonomic nervous system.

It also seems to me that we are running a risk if we venture too far in the field of psychology. It is well known that if we remove any cause of local irritation it may clear up psychic conditions. That is as true of the rectum as it is of the nose. I only wish to sound a warning against carrying too far any particular line of therapy. Some endocrinologists have made that error and brought deserve censure upon the medical profession.

DR. FAULKNER: When your Chairman asked me about two weeks ago to discuss this subject of Sphenopalatine Neuralgia I told him frankly that I knew nothing about it. This seemed to make him all the more insistent that I should say something. I presume from that that he considers ignorance a commendable qualification for public speaking, and perhaps he may be right.

Now, this subject has interested me a great deal ever since the publication of Dr. Sluder's book and I have looked faithfully for cases presenting this so-called syndrome. I have never liked the term neuralgia as a satisfactory description of any phenomenon. The term properly should indicate pain in a nerve where there is no causal factor discovered, and whether such an idiopathic disturbance in a nerve ever occurs is exceedingly doubtful. As our knowledge of disease increases, neuralgias like neuroses tend to become more and more absolute terms. For instance, there is no doubt that in time past the pain in such diseases as appendicitis, pleurisy and even meningitis have been labeled neuralgia, but today we like to have some more specific explanation of any pain manifestation. We have in the posterior portion of the nose a part richly supplied by sensory nerves and it is also a region subject to many pathological conditions which may be rather difficult to determine. It does not seem necessary to me to assume that the sphenopalatine ganglion is such an important factor in producing these various phenomena which we find emanating from this region. I think anatomists have shown quite conclusively that this little ganglion is only a relay station for sympathetic fibers, and nothing more. The description of this automatic mechanism controlled by the sphenopalatine, otic and ciliary ganglion as giving a reflex circuit of sensation and emotional expression in animals is very engenious, but I am afraid he has rather juggled his facts to fit the hypothesis. One might cite the example of a dog manifesting fear from certain sensations of smell which can be changed by presenting some pleasing morsel to him at the same time as he receives the other sensation. His emotional reaction will then change to an expression of pleasure. I suppose the analysis of these sensations is carried out by this wonderful sphenopalatine ganglion. If he can prove this I would advise Dr. Ruskin to take up the study of metaphysics.

As far as I can judge from my own clinical experience I am inclined to believe that many observers of this sphenopalatine syndrome have made a wrong interpretation of certain pathological phenomena and are indebted somewhat to their imagination for their stated facts. Of the definite pathological findings which I have been able to observe I find a pressure effect between the turbinate and the septum in the posterior part of the nose to be the most common. Others have a definite involvement of the posterior sinuses and this may be very obscure. Cases with pressure get temporary relief from cocain, also cocainizing the posterior region of the nose promotes drainage in the sinus cases and a cell with retained secretion may drain itself and give relief

in many cases. Permanent cure of the pressure cases can be effected by a submucous operation or partial turbinectomy, or both. This also may promote drainage of the sinuses.

In regard to the cocainization of the ganglion, I have never believed that so small an application, viz., one-half of one drop, as originally described by Dr. Sluder, could produce much effect on the ganglion, even if it possessed all the function claimed for it. That amount of cocain is usually lost on the middle turbinate as the applicator is pressed between it and the septum. The alcohol injection I have found very difficult and have believed that it could only very rarely be necessary. I am afraid some of my attempts have been failures, but if the ganglion has the functions Dr. Ruskin ascribes to it I am almost glad that I did not injure it.

DR. KLEPPER: Replying to the remarks of Dr. Hubert, I said that the sensory branches pass by the ganglion without entering into its formation. The sympathetic roots, some go into or through the ganglion; the motor branches are not coming from the ganglion. The ganglion is almost entirely sympathetic (vegetative).

I tried to be as conseravtive as possible in the construction of the ganglion. I pleaded conservatism, when I said that very few of the branches go to the tonsillar plexus (probably one-tenth of its supply). I may add that I condemn injecting the ganglion for removal of tonsils, for nose and antrum operations. We must be conservative and not try to attribute everything to the sphenopalatine ganglion and claim results from the injection; we must encourage further research.

DR. RUSKIN: The question of ganglion disturbances has passed the stage where one can assail the results of careful treatment as psychological cures. The scientific work of Sluder and others has placed these disturbances on a definite footing. No one practicing nasal surgery can afford to profess ignorance of the function of the nasal ganglion, which is so important in the physiology of the nasal mucosa. The natomical abnormalities of the nose which produce pain do so through a nerve mechanism, the most frequent one being the nasal ganglion. An impacting septal ridge produces headache, not by its presence, but by its pressure on some nerve filaments. So we cannot disregard the nerve mechanism in speaking of the benefit derived from correction of an impacting septum.

Dr. Hubert tried to show that the nasal ganglion is no sensory ganglion and has no sensory or trigeminal cells. It is obvious that when I classified the nasal ganglion as one of the autonomic ganglian, we would not expect to find nasal ganglion as one of the autonomic ganglia. However, we find sensory trigeminal and facial fibers passing through and around the sphenopalatine ganglion, which in the gross are not to be differentiated from the ganglion proper and which are affected by treatment of the ganglion.

SECTION ON OTOLOGY.

Regular Meeting, Feb. 11, 1927.

Fracture of the Skull Following a Radical Mastoidectomy Simulating Brain Abscess. Dr. Herman F. Lampe.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. PAGE: It is a very interesting case and I congratulate Dr. Lampe on the good results secured. I had a case of hemorrhage that followed an injury to a child who fell on a grape basket and received a punctured wound which caused a large epidural hemorrhage with signs of pressure. This was relieved by removing the bone and recovery was uninterrupted.

DR. DENCH: I would like to know how all these effects were brought about by an extradural hemorrhage—how, one would say, by pressure on the third nerve.

DR. LAMPE: According to Dr. McDonald the pressure from the blood clot caused a paralysis of the third nerve, with a paresis of the sphincter branch; the dilatation of the pupil resulted from the paresis of the sphincter branch, while the paralysis of the superior and inferior oblique and the internal rectus caused the external strabismus due to the unopposed action of the external rectus. As soon as the pressure was removed all these signs disappeared.

DR. MAYBAUM: Was this a bilateral paralysis?

DR. LAMPE: No, it was only on the right side.

Dr. McDonald described the eye condition as a paralysis of the third nerve and a paresis of the sphincter branch, caused by the pressure from the blood clot.

DR. DENCH said that it was interesting to see how these effects could be brought about by a subdural hemorrhage. The third nerve paralysis can only be explained probably by pressure on the cavernous sinus. The clot must have been very extensive in order to cause paralysis of the third nerve.

DR. LAMPE: As soon as the pressure was removed, the paralysis stopped.

DR. MAYBAUM: It was a peripheral paralysis?

DR. LAMPE: That was Dr. McDonald's explanation.

Case of Temporosphenoidal Lobe Abscess. Dr. Morley T. Smith.

Temporosphenoidal Abscess Complicating O.M.P.C. Dr. Morley T. Smith.

Male, age 17 years, admitted to the Manhattan Eye and Ear Hospital on Nov. 20, 1926, service of Dr. John R. Page, complaining of intense right-sided headache, vertigo, ataxia and pain over the right mastoid area, with discharge from the canal. These symptoms had persisted one week and had gradually increased in intensity and patient had slept but a few hours.

Twelve years ago he had fallen down a fire escape and shortly afterwards he developed an acute mastoiditis, for which he was operated. Since then the ear had discharged at intervals, but he had not been under observation. General health had been good.

On admission the patient appeared acutely ill; he had difficulty in standing, and apparently had intense pain. He heard conversational voice at two feet in the affected ear, no nystagmus noted. There was a profuse foul discharge from the right ear, and a large polyp filled the canal. There was some tenderness and redness over the old scar. The eye grounds were normal and the patient did not show any aphasic signs. Temperature 100, pulse 64, respiration 20.

The day of admission a right radical mastoidectomy was performed. The entire mastoid cavity and middle ear were filled with cholesteatoma and pus. The tegmen was necrotic and on removing it, nests of cholesteatoma were found in the substance of the dura, and just above the antrum there was a mass of dirty granulations on the dura. It was suspected at this time that we probably had intracranial involvement, but there was no pus escaping. The wound was lightly packed and left wide open.

The following dav the temperature, pulse and respiration were unchanged; the patient was more comfortable, headache less severe. Later in the day the patient had projectile vomiting, with recurrence of the headache. White cell count showed a total of 17,000, with 78 per cent polys. Urine normal. Patient quite rational—patient did not show any aphasic signs at any time, as it was a right-sided lesion in a right-handed individual.

The second day after the operation the headaches returned with all their intensity, patient irrational at times. Temperature, pulse and respiration unchanged. Eye grounds normal, fields intact. Slight rigidity of the neck. No ocular palsies. When the dressings were removed from the mastoid cavity pus was seen to trickle through a fistula in the granulations previously mention. The patient was returned to the operating room, a crucial incision

enlarged the dural opening, and then with a pair of spreading forceps the fistula was opened and a quantity of pus and gas escaped under great pressure. Spinal puncture at this time showed a clear fluid with no increase in pressure. The cell count was 9, albumin and globulin present, sugar in small amount. Smear and cultures of the spinal fluid we ernegative.

Following drainage of the abscess the headaches disappeared. The pulse remained between 50 and 60 till the fourth day, after which it assumed a normal rate. The temperature was normal throughout. There was free drainage for seven days, when it gradually diminished and ceased draining on Dec. 10. The drainage used was a small, soft rubber tube and was changed daily. We found that very little pus actually drained through the tube, but daily removal worked well in this particular case because of the fistula that had been established. The cavity was not irrigated nor explored in any manner.

On Dec. 3 lipiodol was put in the cavity by the gravity method and X-ray pictures taken. The cavity at this time was fairly well contracted. The first picture showed a small collection one-half inch above the tegmen. Dec. 11 another picture was taken and showed two circular cavities about one-half inch from the cortex and one-fourth inch above the tegmen. Small circular tracts led inward and upward from these cavities. When the lipiodol was first introduced the drainage was fairly free, but it rapidly diminished after this procedure. Whether due to the natural course of events or due to the lipiodol is an open question. Pictures taken Jan. 15 show lipiodol still present.

Cultures from the brain abscess showed streptococcus mitis and bacillus coli.

After Dec. 10, the patient was free of subjective symptoms and has so remained up to the present. He was discharged from the hospital Dec. 30.

This patient is not presented as a cured brain abscess as it is much too early to make such a claim.

DR. PAGE: It was a very beautiful result. Dr. Law can tell us something more interesting about the X-ray plates than I can say.

DR. LAW: I will take the chance of passing these plates around, but I hope that everyone will be very careful with them for they could not be duplicated. There is not much to be said, for the plates tell the whole story. The lipiodol was placed in and shows very clearly on the plates. The peculiar feature is that it remains there so long. It seems as though it was going to be a permanent fixture; but the fact that the discharge has ceased and the case is improving seems to bear out the claim that cases of abscess which have been injected were greatly benefited. It is possible that the iodin in the lipiodol may have some effect which would make it worth while to use it in the treatment of these cases. It is unfortunate that we cannot get lipiodol into the majority of the cases of this type.

DR. MARVIN JONES: The cavity shown by the plate seems to bear out the contention of some that in cases where the drainage is removed the cavity still remains, and the quick mortality of some of these cases at a later date might be explained by the presence of pus, which was not entirely eliminated.

DR. SMITH: The lipiodol was not injected but went in by gravity. Of course about that time the abscess cavity had largely contracted, for there is a very small quantity of lipiodol; the sinus was kept open until there was no more discharge, for many days after the discharge ceased.

Erysipelas Following Pyocyaneus Infection in Mastoid Wounds. Dr. Ralph Armour (by invitation).

DISCUSSION.

DR. DENCH: I don't know very much about erysipelas, but can hardly feel that the Doctor's argument is beyond criticism simply because the streptococcus organisms were not isolated during the attack of erysipelas, for we know that pyocyaneus will outgrow almost anything; and the fact that the streptococci were not found in the smear is therefore of little significance. While there is some evidence that occasionally other organisms do cause or are followed by erysipelas, it does not necessarily follow that the organisms isolated

do cause the erysipelas. The fact that this occurs in inoculated animals carries with that the possibility of infection.

While the paper is most valuable and timely, it does not make much difference whether the erysipelas is caused by pyocyaneus or by streptococcus or staphylococcus; for we have an erysipelas and must allow it to run its course. It does not have a bearing on the antitoxins, for most of them have been lauded and have all died a natural death, just as erysipelas dies a natural death. That does not free us of the necessity of paying great attention and tremendous care in erysipelas; but I believe that cases of erysipelas are no more dangerous than any case of streptococcic infection. I think that many cases of erysipelas which we think are due to another organism are simply adventitious occurrences.

A Clinical and Pathological Survey of Otogenic Nerve Paralysis of the Sixth Nerve. Dr. William H. Sears.

The conclusions set forth at this time are based in the main on the study and analysis of 210 cases of sixth nerve paralysis due to aural suppuration.

This aural complication appears under several forms: isolated; combined with affection of other cranial nerves; contralateral and bilateral. It may even recur; that is, the same patient may, after recovering from one attack, have a recurrence from one to twenty years later. Three such were found in this series, and all made a complete recovery from both attacks.

The syndrome of otogenic abducens paralysis is made up of: *a.* suppurative otitis, *b.* severe temporroparietal pain, and *c.* paralysis of the homolateral sixth cranial nerve.

The syndrome appears with associated clinical symptoms of varying degrees. There appears at times a relatively simple form in which otitis, temporoparietal pain and sixth nerve palsy complete the picture; there are no general symptoms and fever is absent or of slight degree, papilledema is not present, nor is there evidence of intracranial complications. Without operation this type may go on to complete healing within a few weeks, or in other cases a simple mastoidectomy is followed by rapid improvement. On the other hand, the disease may appear in the simple form, as stated, and progress to a point where alarming intracranial complications necessitate extensive surgical interference.

Eventually healing follows or less often the end is lethal. Between the first type, in which spontaneous healing results, and the latter, where death ensues in spite of the smost skillful medical and surgical care, there are variations, most of which are a source of anxiety to the surgeon in charge.

To explain even theoretically an affection of the asserian ganglion giving rise to characteristic pain in some part of its peripheral nervous distribution and an associated affection of the abducens nerve inducing paresis or paralysis, necessitates at least a very brief reference to the anatomical parts involved.

The contiguous position of the Gasserian ganglion and sixth nerve is such as to permit an affection of the same lesion and in most instances this seems the logical course; however, it is quite conceivable that the ganglion can be affected by a lesion at the apex, while the nerve is influenced by a separate condition in the posterior fossa, which may or may not result from disease of the middle fossa affecting the ganglion.

Pathological Diagnosis: A number of authors accept the pathologic basis advanced by Gradenigo; a much large group present cases as typical of his syndrome, but make no mention of the pathology involved. Others present a lesion over the pyramid, ranging from a low grade inflammation of the dura to a temporal lobe abscess, but all with extension to the apex and involvement of the Gasserian ganglion and sixth nerve at that point.

In isolated thrombosis of the jugular bulb, involvement of the inferior petrosal sinus and an inflammatory transfer by this means to the nerve is suggested. In lateral sinus thrombosis with cerebral edema and increased intracranial pressure, pressure on the sixth nerve in Dorello's canal or in its basal course up the pons was proposed by two authors.

Seventeen authors concurred in a diagnosis of paralysis due to a toxic neuritis, but without further explanation.

Alt insists that a reflex paralysis is the only explanation for that class of cases, giving no symptoms of a deep-lying inflammation other than abducens paralysis and which otherwise appear without symptoms and disappear quickly, of which he has observed three. Confirmation by demonstration or deduction:

The evidence from this series that in a certain group of cases tympanic infection progressed by cellular extension to the apex of a pyramid, which is pneumatized to a very unusual degree, is given by the histories of eighteen cases which recorded either the finding during surgical operation or at autopsy of a pneumatic petrous bone, with cells which extended to the apex, and contained pus.

The operative reports which record the finding of pus-filled cells at the point of the petrous, and the disappearance of pain, paralysis and any other symptoms following their drainage, seem to prove that in this type the Gasserian ganglion and sixth nerve have been affected by an inflammatory edema or exudate, or circumscribed pachy or leptomeningitis through contiguity to such a cell or cells, whose walls are inflamed but not necrotic and which react rapidly after the removal of obstruction to free drainage.

Recovery from all symptoms, following proper exposure of the dura for inspection and drainage, with the removal of any intervening focus of infection, of this group of cases, which present dural edema, pachymeningitis or extradural abscess over the roof of the pyramid, from which inflammatory reaction extends to the apex, where again the Gasserian ganglion and sixth nerve can be affected by the same lesion, seems to confirm this as another route for extension of infection to the mesial and of the petrous bone.

Increase of intracranial pressure in thrombosis or thrombophlebitis of the jugular bulb, or endocranial sinuses, following ligation of the internal jugular vein and operation on the jugular bulb or lateral sinus; or in the presence of cerebral edema. In sixty-one cases where the pressure of the cerebrospinal fluid was recorded it was increased in over 65 per cent.

The almost immediate relief of paralysis in certain instances following removal of cerebrospinal fluid by lumbar puncture, drainage of the cisternal pons or magna or by temporal decompression and incision of the underlying dura indicated that in certain and in probably a large group of such cases, the paralysis occurs as a result of mechanical pressure at some point of the nerve's basal course.

A resume of a few facts in connection with the cases which ended fatally may be of interest. There were forty-two deaths, the final cause of which, in thirty-one, was given as diffuse meningitis. The pathogenic organism was recorded in twenty-three cases. In eighteen of these the streptococcus alone was noted; it appeared also in three mixed groups, or 91 per cent. The aural infection was acute in twenty-eight cases and an acute exacerbation of a chronic otitis in eight. In thirty-four where the age was noted, there were twenty-five whose age was 30 years or less, fourteen of these were under 15 years. Above 30 years there were six within the age limit of 45, and but three above that; the eldest was 58. Out of a group of twenty-nine, spontaneous opening of the tympanic membrane occurred in eleven, incision was done in seven, sometimes repeated; three neither opened spontanously nor were incised, and eight were cases of chronic otitis.

There were thirteen autopsies, seven of which gave findings relative to the temporal bone. One showed caries of the posterior wall of the pyramid. In the second an abscess cavity deep in the petrous, from which pus had extended, had ruptured through the petrobasilar suture. The third presented a concavity 9 m.m. in depth near the apex of the pyramid. The other four all contained abscess cavities at the apex of the pyramid; sections of all of these bones were made. Of the others, one showed an extradural abscess surrounding the carotid artery at the point of the petrous. Another presented an isolated thrombosis of the superior petrosal sinus. A third revealed an encapsulated abscess in the pons.

The three remaining, aside from the varying findings of the terminal meningitis, gave nothing of direct value to the question in hand, that had not been already seen during extended surgical operation.

DISCUSSION.

DR. DENCH: It is indeed a pleasure to hear and to discuss such an exhaustive paper on this subject. I myself realize how difficult it was to collect the literature on such a large number of cases, particularly in cases of this character. I wish to congratulate Dr. Sears upon the really remarkable paper which he has presented. Fortunately for the audience, the discussion of a paper of this kind leaves very little for the opener of the discussion. The paper is so perfect in every detail and covers the subject so thoroughly that to attempt either to add to or criticize this masterly exposition would be, indeed, useless.

I have seen a comparatively small number of cases of this kind, although perhaps as many as fall under the observation of the average clinical observer. One point I think should be emphasized, and that is that the severity of the symptoms is rather out of proportion to the mortality of the condition. In other words, from the statistics given by Dr. Sears, and those correspond to my experience, the surgeon should be able to assure the patient and his friends that the appearance of these rather alarming symptoms do not necessarily indicate a pathological condition of unusual severity. While the mortality in the reported cases may seem rather high, we must take into consideration that in every instance we have a pathological process of some sort invading the cranial cavity, and if this is borne in mind the appearance of these very alarming symptoms should not cause undue anxiety.

I am very glad that, thanks to Dr. Sears' paper, the old idea that these symptoms must necessarily be due to a suppuration in the apical cells of the pyramid is controverted. Such an hypothesis would mean, in a great many instances, unnecessary and unwarranted interference. The paper of Dr. Sears has done much to demonstrate not only that the cause of disease may be multiple, but also to give certain very valuable suggestions regarding treatment. I have never seen a case of this kind, so far as I remember, complicating a thrombosis of the lateral sinus, although from the anatomical situation of the sixth nerve certain congenital anatomical anomalies in not only one nerve but both might result from an inflammatory process involving the jugular bulb. In none of my cases, so far as I remember, has the lumbar puncture been of the slightest benefit in combatting the diplopia. This, I believe, to be a very important suggestion. The suggestion of simple increased intracranial pressure from any cause accounting for this symptom, was certainly most interesting, and is, I believe, a valuable one. It seems curious, however, that diplopia seldom exists in cases of brain tumor where the intracranial pressure is sufficiently increased to cause changes in the optic nerve.

I would like also to ask Dr. Sears if in any of the cases reported in literature there has been an herpetic eruption over the distribution of the fifth nerve. It would certainly be logical that such a condition would occur after a perineural inflammation of the ganglion. In no case that I have seen do I remember any such eruption.

I am also interested to learn that certain operators have conceived the idea of relieving both pain and the ocular paralysis by stripping of the dura from the apex of the pyramid. At the time of my operations I do not remember that any such procedure had appeared in literature.

I again express my great pleasure in being permitted to discuss the paper of Dr. Sears, and to have had the opportunity of congratulating him on his very excellent monograph.

Dr. Dench further stated that he did not believe in the reflex origin of these cases. He had never seen the condition complicating a labyrinthitis, but could understand easily how it might occur if the labyrinthitis represented the first stage of a meningitis. The anatomical specimen of the sphenoidal sinus which Dr. Sears had thrown on the screen was of great value, in that it showed the possibility of an abducens paralysis following sphenoidal disease. Curiously

enough, he had recently seen a case operated on at another institution in which each sixth nerve was paralyzed, and this occurred after opening the sphenoidal sinus on each side and the posterior ethmoids.

DR. E. D. FRIEDMAN: After listening to the very excellent presentation of Dr. Sears and to the discussion by my former teacher, Dr. Dench, I rise with diffidence in offering my contribution to the subject under discussion; but there are a few points in the paper that specially interested me. Dr. Sears mentioned the fact that occasionally the Gradenigo syndrome occurs on the contralateral side. I have had an opportunity to observe such a case. The patient was a youngster who had been suffering from an acute left-sided otitis. He developed contralateral external rectus paralysis with characteristic pain. The condition progressed and the patient soon exhibited the symptoms of a cavernous sinus thrombosis on the right. He succumbed to a bacterial meningitis. Dr. Maybaum will recall a patient who presented external rectus palsy in association with homolateral lesion of the seventh and eighth nerves. We postulated an osteomyelitis of the petrous bone in the neighborhood of the internal auditory meatus. The case finally terminated as a cerebellar abscess.

The question of pain interested me very much. Dr. Dench's remarks on the absence of herpes deserve consideration. Herpes is so rare in this syndrome that it seemed to me there might be an explanation for the pain other than that of Gasserian lesion or trigeminal irritation. We know that filaments of the sympathetic accompany the carotid artery and the roots of the fifth nerve. Possibly a lesion of the sympathetic fibers may be responsible for the pain observed in the Gradenigo syndrome. We are, of course, familiar with the fact that sympathetic pain occurs in various parts of the body on the basis of a lesion of the visceral efferents (causalgias).

The pneumatization of the petrous bone is important. Fremal believes that the Gradenigo syndrome occurs only in cases with excessive pneumatization of the petrous bone, in which case the infection creeps along from cell to cell until it reaches the apex of the pyramid. He believes that most of these cases occur in children and that the incidence of a Gradenigo syndrome serves as an indication for operation. He advises that a radical mastoid be done and that as much bone be removed as possible. I have seen perhaps half a dozen cases of the Gradenigo syndrome in my life, but I have not noted its predominance in young individuals. That many recover without operation has already been stated.

One must agree with what Dr. Dench and Dr. Sears have said regarding the nature of the process. I do not think it is toxic. We are dealing in these cases with a gross pathological condition, lepto or pachymeningitis, at the apex of the petrous pyramid. Nor do we deem the paralysis reflex in origin.

Now as to the vulnerability of the sixth nerve, it is well known that the sixth nerve is exceedingly vulnerable. This may be because of its long course, or its relation to the cerebellar arteries which Dr. Sears has alluded to. Involvement of the sixth has no localizing value. Apparently this vulnerability of the sixth nerve demonstrates itself in diverse ways, many of which Dr. Sears dramatically and accurately brought out. Dr. Sears will be interested to know that Kümmel and Brunner are in accord with him in everything that he has said. One could look far and wide for a better presentation of this subject than we have had the pleasure of listening to this evening.

One word more about the case of bilateral external rectus palsy that Dr. Sears spoke of. We have in the hospital, at present, a case of a bilateral sixth nerve paralysis, probably due to a plastic exudate around the sixth nerve. The palsy followed sinus operation. I do not know whether this is the case to which Dr. Dench referred. The slides shown by Dr. Sears are illuminating in this regard. From the standpoint of the neurologist, I should say that such a paper as Dr. Sears has presented demonstrates the desirability of closer co-operation between the neurologist and the otologist in order to further develop our knowledge in the field of neuro-otology.

DR. SEARS: I wish to thank you for your very kind reception of my paper and Dr. Dench especially for his courteous and careful discussion, which has

added very much to the value of the evening. Insofar as my recollection serves he was the first to devise and carry out the surgical procedure which acted so favorably in the case reported.

I was pleased to hear Dr. Friedman's discussion from the standpoint of the neurologist, for I think we all have the same feeling that when the neurologist speaks of the intimate details of otoneurology we who are simply otologists are glad to sit back and listen. He mentioned the vulnerability of the sixth nerve. Some years ago Dr. Dench, in discussion, called attention to the fact that many times an affection of the sixth nerve must be due to its long and exposed course at the base of the brain. I have wondered at times as to whether there may be other conditions which tend to make the nerve more susceptible to attack.

With reference to Dr. Kopetzky's discussion on treatment of patients showing the radenigo syndrome, in the main I am quite in accord. As has been noted before, Dr. Kopetzky in his recent work on surgery of the ear covers the radenigo syndrome in a concise, clean-cut and more clearly expressed way than I have seen any place else. The question under discussion this evening, however, is that of otogenic paralysis of the abducens nerve, of which the Gradenigo syndrome forms one part, and the discussion is limited to symptoms and pathology.

The appearance of the symptoms complex of abducens paralysis in suppurative disease of the middle ear is relatively rare. This is equally true of its intracranial complications.

Dr. Perkins collected 102 cases of otitic meningitis, in which he found this symptom recorded four times; in more than 100 cases of sinus thrombosis recorded in the literature, it was noted sixteen times; in extradural abscess of the middle or posterior fossa its appearance is infrequent; in temporal lobe and cerebellar abscess it is seen but rarely.

In some of the cases presenting the Gradenigo syndrome, of which Dr. Kopetzky has spoken, the patients get well without any surgical operation and we have no way of proving the pathology involved, but why do they not occur more frequently? Again, why in cases of otitic meningitis with increased intracranial pressure, not in the terminal stage but during its course; or in the majority of extradural abscesses and in the cases in which you find your tegmen tympani or antri affected, is it not more frequent?

This problem and its solution I would like to leave with the Section. We can have extension of tympanic infection through the pyramid, over the pyramid, by way of the mastoid and by other routes, resulting in the production of this symptom complex, and yet as a matter of fact its appearance is relatively infrequent. Why?

The American Board of Otolaryngology conducted an examination at Washington, D. C., on May 16 and 17, and at Spokane, Washington, on June 4. Of the 142 men examined at Washington, D. C., 119 were passed and 23 failed to pass the examination. In Spokane, the number passed was 46, and the number failed was 6.

The next examination will be held in Detroit on September 12, 1927. The applications for examination should be sent to Dr. H. W. Loeb, Secretary, 1402 South Grand Boulevard, St. Louis, Missouri.